Structural Violence in Éire: The Bone Histology of Victims from the Great Famine (Kilkenny, Ireland 1845-1852)

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Abstract

This project uses bone histological analysis to explore how disease, diet, and social circumstances affected the bone remodeling of a group of people who died during the Great Famine in Ireland between 1845 and 1852. The human remains discovered on the grounds of the nineteenth century Kilkenny Union Workhouse in Kilkenny City, Ireland represent a well contextualized, homogenous group of people who died due to stress induced by the Great Famine sometime between 1847-1851. These factors make this an ideal population to study the biological effects of structural violence and for investigating the meaning of skeletal lesions, often referred to as the "osteological paradox".

In 1845, a potato blight wiped out the primary means of subsistence for the poor in Ireland, causing the country to lose a quarter of its population to death and migration. This event had a massive cultural, demographic, and biological impact on the world and has been the source of international scholarly interest in the recent past. Historical research has suggested the poor in Ireland suffered and died from comorbidities of infectious and metabolic disease due to food insecurity, the cause of which is debated but is often attributed to conflicts with England. Bioarchaeologists have recently examined the effects of diet and disease on the macroscopic surface of the skeletons but none have looked at the effects of the Great Famine on their bone microstructure.

The skeleton reacts to physiological effects of stress on the micro level before presenting as lesions on bone, and since many diseases are not reflected in the bioarchaeological record, histological analyses of bone may be informative of the lived experience of those with and without lesions. This study compares bone remodeling patterns in the ribs of 99 adults and 87 subadults between four disease categories: metabolic disease, infectious disease, comorbidities of infectious and metabolic disease, and no lesions, to determine if variation in bone microstructure exists between disease types. This was conducted using bone histological variables that reflect the maturity of remodeling and extent of porosity, which can be indicative of overall health. Additionally, carbon ($\delta^{13}C$) and nitrogen ($\delta^{15}N$) stable isotope values obtained from the ribs of the same individuals were compared to bone histological variables to determine if changes in diet due to the loss of the potato and the introduction of maize as a relief food influenced bone remodeling.

The results show that rib bone porosity is highest in adults without lesions and lowest in adults with evidence of infectious disease. Similar results were obtained in the subadult cohort. Additionally, those with evidence of scurvy generally show less porosity throughout the rib cortex for both age cohorts. Comparisons of histomorphometry with stable isotope values showed a positive correlation between osteon size and $\delta^{15}N$ values in adults as well as a positive correlation between Haversian canal size and $\delta^{13}C$ values in the subadults, possibly indicating slower remodeling in those with more evidence for starvation and less mature remodeling in those with maize in their diet, respectively. This study showed that bone histology is impacted by both disease and diet and may be useful for interpreting the meaning of lesions and understanding the impact of social status on population health.

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This thesis is dedicated to those who lost their lives during the Great Famine.

CHAPTER 1: A HISTOLOGICAL ANALYSIS OF THE GREAT FAMINE (1845-1852)

Human health is a complex state dictated by forces acting from both within and outside the body. An important aspect of our life experience, health is characterized by various types of well-being including individual perception and the physiological and psychological conditions of well-being (Temple and Goodman 2014; Logan and Jacka 2014). For many of the 8.6% of the world's population who live in extreme poverty (generally defined as living on less than US$2 per day), achieving a state of idealized homeostatic well-being over the lifetime is unattainable due to structural violence, or the social, cultural, and economic circumstances that restrict access to resources (UN Global Sustainable Development Report 2019).

Deviation from a homeostatic state of health is often referred to in biomedical literature as "stress" (Temple and Goodman 2014). The experiences of stress are not only transiently embodied during life, but some forms of stress remain recorded in the skeleton after death. Bioarchaeologists are trained to examine human remains in order to observe patterns of health and illness over the course of human history, which can inform us of the life experiences of those who have suffered from structural violence. This study examines bone morphology at the microscopic level to understand the consequences of disease, malnutrition, and social inequality for skeletal health within an impoverished community who died during the Great Famine in Ireland (1845-1852).

The potato blight (*Phytophthora infestans*, an airborne fungus) that initiated the Great Famine arrived in Ireland in 1845 and subsequently reduced the population by over two million people (Mokyr 1983). However, it was not just an enormous number of people that were lost; an equally powerful outcome of such a large demographic shift was the waning of traditional Irish culture, specifically the Irish Gaelic language and rural folklife, as well as the physical and mental well-being of the people who survived the Famine and remained in Ireland (Mokyr and Gráda 1982; Kelly 2019). Of those experiencing extreme levels of poverty and deprived living conditions today, 85% live in regions such as Syria, Afghanistan, and Iraq where conflict and resource insecurity such as famine are affecting their daily lives (UN Global Sustainable Development Report, 2019).

The uninhabitable circumstances created by these crises is a primary force driving migration with international consequences as refugees seek asylum in countries all around the world (UN Global Sustainable Development Report 2019). While the impetus for migration varies depending on regional situations, for millennia human movement has been initiated by life-threatening circumstances of poverty, violence, and climate change—each of which leads to food insecurity as well as other circumstances that cause poor health if the harmful situation does not change or new environments are not sought out (UN Global Sustainable Development Report 2019). This was the ultimatum for the emigrants who left Ireland during the Great Famine in the middle of the nineteenth century. For those who were able to travel to England, North America, or Australia, escaping was a matter of life or death.

Recently, the written history of the Great Famine, which is mostly constructed from archival documents, travel accounts from numerous visitors to Ireland, and folklore, has been supplemented with archaeological and bioarchaeological materials from mass burials discovered at the site of the former union workhouse in Kilkenny City, Co. Kilkenny. The former Kilkenny Union Workhouse was used to bury victims of starvation-induced disease and malnutrition beginning at the height of the Famine in August 1847 and was used until March 1851 (Geber 2015). Previous bioarchaeological studies from this time population have revealed aspects of the difficult lives these people endured prior to and throughout the Famine (Geber and Murphy 2012; Beaumont et al. 2013; Beaumont and Montgomery 2016; Geber et al. 2019; Geber and O'Donnabhain 2020).

This thesis shows how combining previously collected bioarchaeological data with microscopic evidence of stress experiences that were recorded in the bones during life, also known as bone histology, can contribute to the understanding of the consequences of disease and diet (nutrition). In the past, bioarchaeological studies of human health and stress have utilized various lines of evidence including paleopathological analysis of lesions on the skeleton, the observation of imbalances in bone cellular remodeling, and chemical (stable isotopic) evidence of diet. In these studies, observations of macroscopic and microscopic stress are often linked to metabolic and infectious diseases brought on by malnutrition, poor water quality, and unsanitary living conditions (Larsen 2018; Brickley, Ives, and Mays 2020b); while light stable isotopes have informed us about the dietary habits that contribute to health, or lack thereof

(Arnay-de-la-Rosa et al. 2011). In this thesis, the quantification of bone histology, known as bone histomorphometry, contributed to the understanding of the consequences of disease and diet (nutrition) of those who died during the Great Famine and were discovered on the grounds of the former union workhouse in Kilkenny City.

The Great Famine is known as one of the largest social catastrophes of the nineteenth century because it affected an entire social class who relied exclusively on the potato for subsistence. When one crop is cultivated on the same land for multiple seasons it is called a monoculture (Power and Follett 1987). Monocultures are dangerous for several reasons, including the lack of nutritional diversity and the dangers of seasonal famines due to crop failure from environmental circumstances or pathogenic invasions. Dietary diversity is important for regulating the metabolic functions necessary for maintaining soft and hard tissues. For example, collagen is necessary to maintain the integrity of connective tissues in and around the blood vessels; when Vitamin C is deficient, collagen cannot be synthesized (Blee et al. 2002), blood vessels become weak, and hemorrhaging of skin, muscles, and beneath the surface of the bone may occur (Aghajanian et al. 2015). In the latter case, skeletal lesions may form that remain identifiable and pathognomonic for scurvy after death (Ortner et al. 1999).

While some diseases, namely scurvy, rickets/osteomalacia, and iron deficiency anemia, can be identified in the archaeological record, diagnosis is complicated by the fact that many diseases leave similar traces on the skeleton and individuals can suffer from more than one disease at the same time (Ortner et al. 1999). For example, Vitamin C deficiency commonly occurs alongside iron deficiency anemia and may produce osseous changes in line with symptoms of pellagra (Ortner 1991; Armelagos et al. 2014; Klaus 2017). Additionally, the absence of lesions does not necessarily indicate the absence of disease, since diseases often will never form hard tissue lesions (Ortner and Aufderheide 1991) and lesions are less likely to form in adults not experiencing continuous growth (Armelagos et al. 2014). These concerns address what is known as the "osteological paradox" (Wood et al. 1992). While bone histology is not diagnostic of specific diseases, it can provide a foundation for understanding the cellular processes that are occurring beneath the surface of the bone. For example, since collagen is an essential component of bone formation, when there is a lack of Vitamin C the bone loss and osteoporosis that may occur can be observed as porosity in bone histological analyses (Herrmann et al. 2007).

The relationship between skeletal pathology, disease, and diet has been studied in skeletal populations to better understand the effects of lifestyle, social status, and the culture of people from the past. Since skeletal lesions, osteoporosis, and other effects of physiological stress develop from the actions or inactions of bone cells in response to metabolic changes, bone histology may be informative of the presence of physiological disturbances before lesions develop. Specifically, histological analyses of the ribs and femur have shown evidence of stress in populations suffering from poverty and disease in the years leading up to death. For example, a study by Martin and colleagues found that Black adults who died just after the reconstruction period in America (1878-1930), when the Civil war had ended but racial segregation was still protected by the law, suffered from poor nutrition that was reflected in their rib bone microstructure (Martin, Magennis, and Rose 1987). Similarly, a more recent study revealed how poverty and social conditions during the Apartheid era in South Africa (1948-early 1990s), a time of violent, institutionalized racial segregation, can influence changes in adult rib bone histomorphometry for those who suffered from the nutritional deficiency disease pellagra (Brenton and Paine 2007). Robbins Schug and Goldman (2014) found young children with shorter stature and fewer paleopathological lesions than their taller peers did not maintain or acquire the bone mass expected for their age. For this group, bone histological analysis showed that conventional ideas about the presence of gross lesions should be critically evaluated.

The nutritional status of archaeological populations is intricately linked to disease. To better understand the etiology of disease in a population, the diets of past people can be inferred through several methods, including botanical and faunal samples, dental calculus, and most importantly for this study, historical records and stable isotope analysis. Stable isotope analysis is the investigation of the chemical components of bones and teeth that reflect individual diet just before death or in childhood, respectively (Katzenberg and Water-Rist 2019). For example, analysis of carbon isotope values ($\delta^{13}C$) from the collagen of ribs can indicate whether maize was consumed in the years leading up to death and nitrogen values ($\delta^{15}N$) may indicate nutritional deficiencies (Buikstra and Milner 1991; Beaumont et al. 2013).

The Kilkenny Union Workhouse skeletal population is derived from a region with vast historical documentation of the catastrophic event that led to the death of those who were buried there. This provides a unique opportunity to investigate the biological consequences of low

social status on the health of a marginalized and impoverished population. For example, Jonny Geber found high frequencies of skeletal pathological changes indicative of metabolic and infectious disease in the workhouse population (Geber 2015). The presence of pathological changes on these remains means that there was a disruption in the homeostasis of normal cellular processes which was extended enough to create osseous lesions. Additionally, Beaumont and colleagues found lower $\delta^{15}N$ values in the Kilkenny sample relative to other contemporaneous populations known to have better health (Beaumont et al. 2013). They also documented evidence of $\delta^{13}C$ values that indicate maize consumption, which complements historical literature describing the distribution of maize as a relief food to inmates of the workhouse during the Great Famine (described in more detail in Chapter 2) (Beaumont et al. 2013).

Since evidence of disease and nutritional status can be reflected in the rib bone microstructure prior to the appearance of lesions on the external surface of the bone (Frost, 1963), histomorphometric analysis of the ribs can show new evidence of the quality of bone health for those who were suffering during the Great Famine, and reveal whether their bone maintenance was improved by the introduction of maize, the relief food provided by the British Government. This study measures standardized morphological features of bone microstructure (the amount of cortical bone, bone porosity, osteon and Haversian canal size and shape, osteon population density, and frequency of double zonal osteons) to understand relationships between disease and dietary evidence identified in the skeletons from the Kilkenny Union Workhouse to further characterize the experience of those who lived prior to and during the Great Famine.

1.2. AIMS, OBJECTIVES, AND HYPOTHESES

The examination of a historical case of extreme poverty where migration, resigning to the workhouse, or death were the only options for most people may help explain the decisions modern populations face while suffering from social conditions outside their control, such lack of access to a diverse diet. These conditions are the result of what is referred to as structural violence, a phenomenon that reinforces damaging socioeconomic hierarchies by restricting access to resources for those at the lower levels of society (Galtung 1969). Structural violence places disenfranchised groups in vulnerable situations that often lead to poor individual and group health outcomes, such as systemic poverty, which increases the risk of death. Historical

records describing the living conditions and experiences of the poor in nineteenth century Ireland are evidence of structure violence that results in poor bone health evident by the presence of macroscopic lesions observed by Geber and colleagues and microscopic features described in this thesis (Geber and Murphy 2012; Geber 2015).

While much research has been conducted on the history of the Famine and recent paleopathological analysis has explored the expression of disease on the skeletons of those who were buried on the grounds of the Kilkenny Union Workhouse, no study yet has looked deeper into the bones to explore how the workhouse inmates were experiencing stress on a microscopic level. This thesis is the first bone histological study on historically contextualized skeletons from Ireland, the first that relates to a population that suffered through a well-recorded, long-term famine, and an important contribution to the understanding of the response to malnutrition and disease in a marginalized community.

AIM OF RESEARCH: The aim of this research is to quantify histological indicators of stress due to disease and diet to better understand the experience of the men, women, and children who died and were buried on the grounds of the Kilkenny Union Workhouse during the Great Famine in Ireland. Histological indicators of stress may manifest as an imbalance in bone remodeling represented as decreased percent cortical area, increased pore size, increased percent porosity, low osteon population density, enlarged osteon and Haversian canal size and shape, and high frequencies of double zonal osteons. The aim is achieved through a series of research questions and hypotheses:

Question 1: *Is there a difference in the rib histomorphometry of individuals that display lesions indicative of disease and individuals without lesions?* The first goal is to investigate how bone responds to disease on a microscopic level. This was accomplished by documenting key histomorphometric variables in the cross-section of the mid-rib to observe how disease impacts bone remodeling. These variables were compared to the macroscopic paleopathological analyses conducted by Geber (2015) on the Kilkenny Union Workhouse population to determine if expressions of histomorphometry vary with expressions of osseous lesions (pathological changes in the skeleton) including those with only infectious disease, only metabolic disease, both metabolic and infectious disease, or without lesions. The study will

explored the relationship between lesion presence and histomorphometry to untangle the osteological paradox and the identification of "frail" individuals within the Kilkenny Union Workhouse population. The null hypotheses are as follows:

Null hypothesis 1.1: *There is no difference in the rib histomorphometry of skeletons that display macroscopic lesions indicative of metabolic disease and individuals without macroscopic evidence of metabolic disease.*

Null hypothesis 1.2: *There is no difference in the rib histomorphometry of skeletons that display macroscopic lesions indicative of infectious disease and individuals without macroscopic evidence of infectious disease.*

Null hypothesis 1.3: *There is no difference in the rib histomorphometry of skeletons that display macroscopic lesions indicative of both metabolic and infectious disease and individuals with macroscopic evidence of only metabolic disease, only infectious disease, and without lesions.*

Null hypothesis 1.4: *There is no difference in the rib histomorphometry of skeletons that do not have macroscopic pathological lesions and individuals with evidence of macroscopically identified disease lesions.*

Question 2: *Did relief food impact the histomorphometry of the individuals buried in the Kilkenny Union Workhouse mass burial ground?* The second aim of this research is to reveal changes in rib bone histomorphometry in response to changes in diet. Those who entered the workhouse institutions were the most vulnerable in society who depended on relief food to survive. Bone histological analysis can inform the state of individual health with and without the presence of relief food by investigating how the remodeling process of the rib is impacted by levels of light stable isotope values. To achieve this aim, rib histomorphometry will be compared to the stable isotopes values of carbon ($\delta^{13}C$) and nitrogen ($\delta^{15}N$) which were obtained from the same rib samples (Beaumont et al. 2013). Beaumont and colleagues showed evidence that the relief food maize (a C_4 plant), indicated from high carbon values (>-17.0‰), has been incorporated into the rib collagen for some samples (Beaumont et al. 2013). If there are correlations between bone histology and C_4 values, then the results of this analysis will reveal how bone remodeling responds to the introduction of maize during a period of severe

malnutrition in a sample of impoverished individuals dependent on relief food for subsistence. If there are correlations between bone histology and $\delta^{15}N$ values hypothesized to be indicative of malnutrition (> 12‰) then the impact of starvation on bone remodeling can be assessed.

Null hypothesis 2.1: *Changes in diet and severe nutritional stress did not affect the rib histomorphometry*. This hypothesis will be tested by comparing histomorphometric data obtained from the rib between:

a. $\delta^{13}C$ isotope values indicative of relief food consumption (values higher than -17‰)
b. $\delta^{15}N$ isotope values indicative of malnutrition (values higher than 12‰)

1.3. CHAPTER LAYOUT

Chapter 2 includes an overview of bone biology including descriptions of the functions of bone cells and the process of skeletal growth and development. The types of human bone are also described and the application of bone biology in the field of bioarchaeology is discussed. This chapter also introduces the biocultural approach to bioarchaeological research to set up the themes explored in this thesis.

Chapter 3 provides a brief history of the events leading up to and throughout the period of the Great Famine within the framework of structural violence to contextualize this sample within its cultural and sociopolitical spheres.

The materials and methods used in this thesis are described in Chapter 4. Here, the method for construction of the skeletal sample is explained and the process of bone preparation is outlined. Then, the disease type categories are described, an overview of the values obtained from stable isotope analysis are listed, and the statistical analyses used for disease and isotope comparisons are presented.

Chapter 5 provides the results of the histological analyses used to explore the impact of disease and diet on bone histomorphometry. This chapter is divided into disease types to present the first question and statistically significant data for the adult and subadult cohorts are included for each section.

Chapter 6 is discussion of the results of the relationship between disease and diet and bone histology in the context of the Great Famine. This chapter aims to show how variation in bone histological features can be used to better understand the experience of disease and diet for the victims of the Great Famine, how this information can provide greater detail about the meaning of lesions in bioarchaeological samples with regard to the osteological paradox, and to provide more context for the biological consequences of structural violence today.

The final chapter concludes the discussion on the bone histology and the Great Famine with an overview of the research discussed in this thesis, considerations for future work, and the impact this research has on the field of bioarchaeology.

CHAPTER 2: BONE BIOLOGY IN BIOARCHAEOLOGICAL CONTEXTS

The strength, endurance, and significance of the skeleton can be observed on a macro, micro, and nano scale where bone, a hierarchical element, is reacting to its environment at every level (Ruppel et al. 2008; Martin et al. 2015). Histology, or microscopic anatomy, is the observation of tissues at the intermediary level (Stout and Crowder 2012). In biological anthropology, bone histological analysis involves the microscopic examination of human bone to observe the patterns of cellular activity that form, remove, and replace skeletal tissue while maintaining skeletal health. Without a consistent balance of this activity, bone can become fragile or more robust. Bone fragility, whether through porosity or hypo-mineralization, can cause fractures that lead to pain, immobility and require energy to repair (Seeman and Delmas 2006; Ruppel et al. 2008). In this chapter, the skeleton will be introduced at the histological level to explain how growth and development, behavior, and diet affect the skeleton's ability to adapt and maintain its structural properties. First, the basic anatomy of the skeleton will be introduced. Then, a discussion of the histological features of bone and how they are affected by disease and diet will occupy much of this section as it is the focus of this thesis. Finally, an overview of the utility of bone histology in bioarchaeology will be provided. This is relevant for understanding how the life experience of disease and malnutrition affected the bone microstructure of the people who were buried on the grounds of the Kilkenny Union Workhouse.

2.1 INTRODUCTION TO BONE

The skeleton is a dynamic organ that serves multiple functions—it acts as a lever during mobility and provides support of soft tissue, protects the vital organs, produces red blood cells in the marrow, and stores nutrients such as calcium and other minerals throughout the body (Burr 2011). Functionally, the shape and structure of the skeleton is dictated by the coupled action of bone cells that replace old bone with new bone during growth, development, and biomechanical loading. However, the functionality of these bone cells is dependent on reliable metabolic processes which can be affected by external factors such as diet and disease (Agarwal and Miller 2016). By interpreting the patterns leftover from the actions of these cells, bioarchaeologists can use the skeleton to understand the experience (and concept) of health in past human populations (Agarwal and Miller 2016; Miszkiewicz and Cooke 2019).

The skeleton can be divided into three different types of bones based on their morphology: long bones, flat bones, and irregular bones. Long bones include the humerus, ulna, radius, femur, tibia, and fibula; flat bones are most bones of the skull, sternum, ribs, and scapula; and irregular bones are those of the pelvis, carpals, tarsals, and vertebrae (Buckwalter et al. 1995). Each is covered by a thin membrane called the periosteum which lines the dense outer portion of bone referred to as cortical bone or the cortex. Inside the cortex is a medullary cavity which is composed of fatty yellow marrow and spongy bone called trabecular or cancellous bone. Trabecular bone is made up of a web of rods and plates to form an interconnected network of bone that houses red marrow and produces and stores red blood cells. A membrane called the endosteum separates the cortical bone from the medullary cavity (Rho et al. 1998).

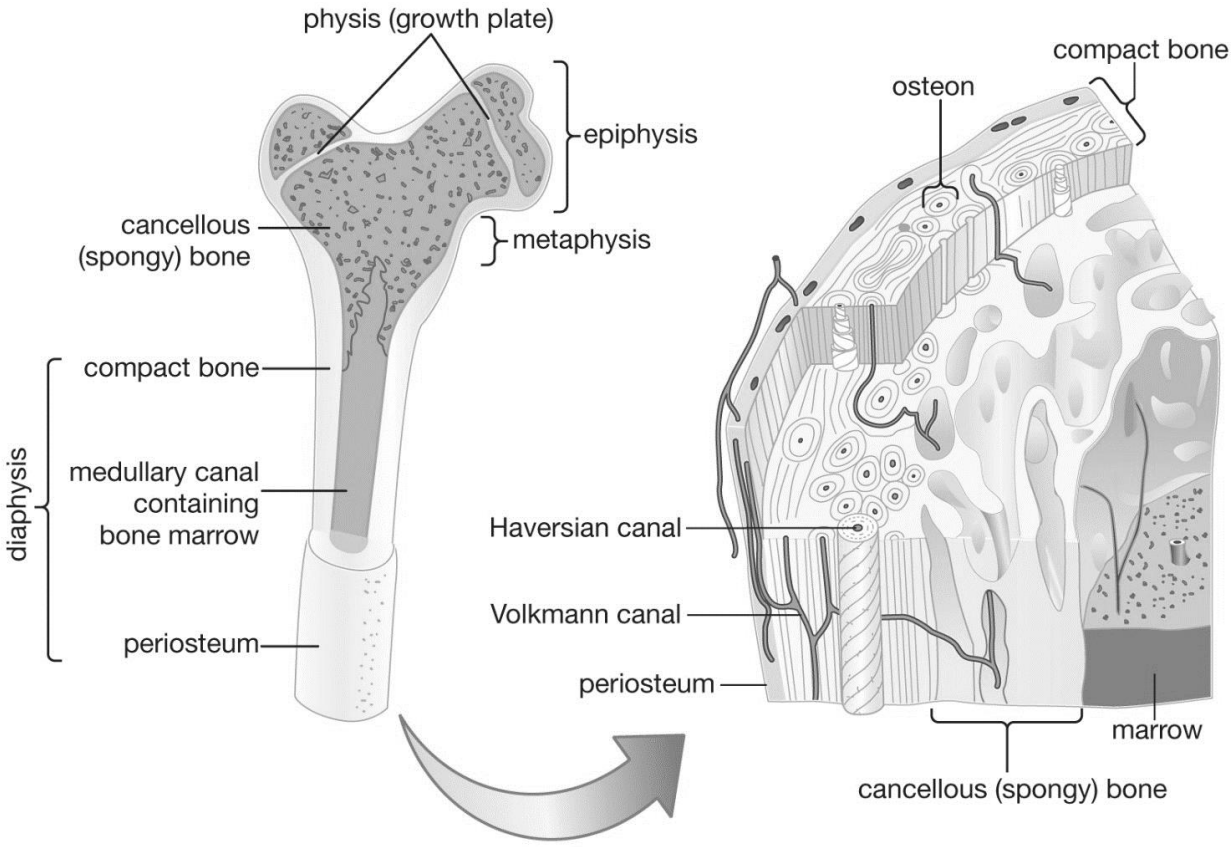


Figure 2.1. Macro- and microstructure of bone. Source: Adapted from Merriam-Webster (2012)

Within the cortical and trabecular bone are systems of cellular activity that regulate bone growth, development, and repair throughout life. For these systems to function properly they are dependent on adequate bioavailability of minerals that aid in bone modeling and remodeling as discussed in this chapter (Cashman 2002). In populations where dietary resources are restricted and people are not receiving adequate nutritional intake, such as in countries experiencing conflict or natural disaster, these regulatory metabolic systems are disrupted causing bone mineral reservoirs to be depleted and bone to become weak under its specific loading environment. Not only can impaired bone strength lead to increased micro-damage, fracture, infection, and immobility (Parfitt 1993; Cashman 2002; Brickley, Ives, and Mays 2020b), but nutritional deficiency compromises the immune system and co-occurrence of disease often proliferates metabolic instability, leading to more complex disruptions in bone health (Brickley, Ives, and Mays 2020a).

Bone histomorphology is the term used for the identification of microscopic features in bone, while histomorphometry is the term used for the quantification of bone cell activity (Eriksen et al. 1994; Pfeiffer 1998). Since histomorphometric analysis involves measuring the features of bone that can be affected by the life experiences mentioned above, it is important to understand the process of their ontogeny to answer the questions presented in this thesis. This section describes the basic bone microanatomy including osteoclasts, osteoblasts, osteocytes and bone-lining cells, all essential for maintaining bone metabolism, growth, and adaptation. Then, the processes of intramembranous and endochondral ossification are described and the significance of bone modeling and remodeling for the maintenance of bone health are explained. Finally, cortical bone types and the anatomy of Haversian systems (osteons) are detailed, as well as other bone histology variables involved in the metabolic process, such as resorption bays. The cortex of the rib, a flat bone, is of primary interest in this thesis for theoretical and practical reasons that will be described in this section.

2.2. BONE CELLS

This section describes the intermediate histological features of cortical bone of the skeleton where, in the case of well-preserved specimens, the remains of bone cell activity can be observed as they were at the time of death. Throughout life, mechanical and physiological demands are the driving force behind the activation of cells that change bone size, shape, and

mass (Ruff et al. 1992; Frost 1988). While growth and development can occur at a consistent rate in groups that maintain nutritional and biomechanical standards, disruptions to homeostasis due to physical activity, diet, or disease can change the characteristics of bone in isolated or generalized regions (Simmons 1990) causing lesions such as those observed in the Kilkenny Union Workhouse population sample (Geber 2015) (See Section 3.2.1). Biological and genetic variation can also dictate the differing response of bone cells to life events such as famine or other changes in subsistence. These differences can exist between and within populations, causing some individuals to be more susceptible to disease than others. This concept, known as heterogeneity in frailty, is discussed in Chapter 3 (Frost 1987; Stout and Lueck 1995).

To understand how histomorphological variables are affected by disease processes, it is important to comprehend the basic cellular mechanisms by which they are formed and the various ways in which normal metabolic maintenance can be disrupted. The following section describes the primary bone cells that form the histomorphological variables under investigation in this thesis and are responsible for the process of bone maintenance, including osteoclasts, osteoblasts, osteocytes, and bone-lining cells.

2.2.1. OSTEOCLASTS

The homeostatic balance of bone for mineral transfer and biomechanical repair is dependent on the skeleton's ability to maintain bone turnover through the removal of old bone that is subsequently replaced with new bone. The process of removal is achieved by the osteocytic activation of osteoclasts, multinucleated bone-resorbing cells that degrade bone by secreting proteolytic enzymes and acid (Charles and Aliprantis 2014). These cells are the product of osteoclast precursor cells which start in bone marrow as hematopoietic stem cells and differentiate into precursor cells by cytokine colony stimulating factor CSF-1 and receptor activator RANKL which has been produced by osteoblasts and osteocytes (Teti 2013; Charles and Aliprantis 2014). Osteoclasts also play a large role in the activation of osteoblasts in balanced remodeling (Parfitt 1982). This balance is called "coupling". It is hypothesized that in the act of bone resorption osteoclasts secrete clastokines, molecules that contribute to the regulation of bone formation by osteoblasts (Teti 2013). If the secretion of clastokines is somehow inhibited, osteoblast activity will not follow bone resorption, causing an imbalance

in the coupling process that can lead to osteopenia and later osteoporosis (Cashman 2002; Teti 2013).

Under the microscope, dry bone presents features such as a resorption bay that represent regions where osteoclasts would be found in life. Resorption bays are large areas of porosity within the cortical bone that represent remodeling sites (Sedlin, Villanueva, and Frost 1963; Maggiano 2012). In life, osteoclasts are present in depressions of the bay that have a distinct "ruffled border", also known as a Howship's lacuna (Arnett and Orriss 2018). The presence of osteoclast induced Howship's lacunae throughout the cortex is normal, as they are necessary for bone remodeling to occur; however, they must be constantly filled in by osteoblasts. If osteoblasts are not actively secreting osteoid for bone formation, then the gaps in the cortical bone will remain, causing hazardous levels of porosity that contribute to the potential for bone fracture and inhibit oxygen and nutrition distribution throughout the bone (Stout, Cole, and Agnew 2019). For example, in individuals with hyperparathyroidism, dietary calcium deficiency causes excessive parathyroid hormone, which induces osteoclastogenesis leading to increased bone resorption and osteoblast deposition of weak, under-mineralized woven bone (McCarthy 2016; Stout, Cole, and Agnew 2019).

2.2.2. OSTEOBLASTS

Osteoblasts are the bone-forming cells essential to both the modeling and remodeling process. Osteoblasts form from alkaline-phosphatase positive cells known as preosteoblasts, which are active progenitor cells that differentiate from mesenchymal stem cells (Charles and Aliprantis 2013). In modeling and remodeling, osteoblast activity is initiated by chondrocytes or osteocytes respectively, which determine the need for bone building in response to mechanical or physiological demands (Gosman, Stout, and Larsen 2011).

During modeling, cartilage is removed by chondroclasts while osteoblasts lay down a matrix of osteoid composed of Type I collagen fibers, proteoglycans, water, and other non-collagenous proteins. Mineralization of osteoid, which is necessary for bone to form, does not occur until about 10 days after initial deposition but mineralization can be disrupted if the amount of calcium phosphate that is produced by osteoblasts and deposited into the matrix is not properly regulated (Allen and Burr 2014b). Regulators for osteoblast mineralization are inorganic

pyrophosphates which inhibit the abnormal calcification of bone and the protein osteopontin. This mineralization component is important for the maintenance of bone strength and the continued process of bone remodeling since osteoclasts cannot perform their job if osteoid has not been mineralized (Stout and Crowder 2012). After depositing osteoid, osteoblasts undergo apoptosis in one of two ways—they either become incorporated into the under-mineralized matrix to become osteocytes, or, if they are still present at the cessation of formation, they become bone lining cells. Bone lining cells (described later in this section) remain on the bone surface where they can be reactivated by osteocytes to begin the osteoid deposition process again (Franz-Odendaal, Hall, and Witten 2006).

Throughout senescence or when trauma or pathology has affected the bone, osteoblast activity can slow down or fail to initiate mineralization, which uncouples the remodeling process and increases intracortical porosity and the risk of fracture. Evidence of Vitamin C deficiency, known as scurvy, was more frequently identified in the Kilkenny Union Workhouse skeletal sample than any other disease. In individuals suffering from scurvy, collagen production is limited and; therefore, osteoblasts cannot deposit osteoid. Since osteoclasts continue the process of resorption, this may lead to greater porosity, osteopenia, and osteoporosis which can be identified using bone histological methods (Ortner and Ericksen 1997; Fain 2005; Brickley, Ives, and Mays 2020b). However, the human body only requires a small amount of Vitamin C for collagen formation and associated mineralization to occur, so it is only under circumstances of extreme deprivation for extended periods of time that scurvy will develop (Mays 2014).

The function of osteoblasts is also disrupted by Vitamin D deficiency, referred to as rickets in children or osteomalacia in adults. In the case of Vitamin D deficiency, calcium cannot be absorbed through the small intestine which reduces the amount of bioavailable calcium for osteoid mineralization (McCarthy 2016b). In this state osteoblasts will continue to deposit osteoid, but osteoclasts will not be able to resorb the unmineralized bone. This leads to a thick under-mineralized cortex and osteoid seam that covers most of the bone and cannot be resorbed until Vitamin D has been reintroduced and mineralization can be initiated (Oppenheimer and Snodgrass 1980; Stout and Teitelbaum 1976). When bone is too flexible, as in the case with Vitamin D deficiency due to impaired mineralization, it is prone to fracture even under normal loading circumstances (Seeman and Delmas 2006). Under the microscope individuals with

Vitamin D deficiency may exhibit a separation between the central Haversian system and the surrounding interstitial bone. Large resorption cavities and enlarged osteocyte lacunae are also pervasive, particularly in the ribs and other bones with higher rates of turnover (Brickley, Mays, and Ives 2007; De Boer and Van der Merwe 2016; Stout, Cole, and Agnew 2019). The impact of disease on bone histology is explained in further detail in Section 2.5.

2.2.3. OSTEOCYTES

During bone formation, between 10% and 30% of osteoblasts are embedded into the bone matrix during formation to become osteocytes (Komori 2013). Osteocytes are the most plentiful cells in human bone and serve as the directors of bone remodeling (Bonewald 2011). These cells are responsible for initiation of osteoblast or osteoclast activation in response to paracrine and autocrine factors that sense alterations in the mechanical loading environment (Bonewald 2011; Bell, Kayser, and Jones 2008). Osteocytes rest within lacunae that are connected by long, fluid filled dendrites called canaliculi which allow communication and flow of nutrients between regions of bone. This network, called the lacunar canalicular system (LCS), senses changes due to mechanical stimuli and instigates osteocytes to signal osteoblast activity in areas of high pressure (Main 2017).

Microdamage due to mechanical demand or injury also sets off LCS alerts, activating other osteocytes through the LCS. When local microdamage to bone occurs, canaliculi extend from the lacunae where the osteocyte is housed, providing a channel through which local communication with other osteocytes can occur to instigate modeling and remodeling of the affected environment (Bell et al. 2008). However, extensive microdamage in bone can disrupt signaling between cells and result in osteocyte apoptosis and empty osteocyte lacunae. In this case, bone fragility is increased due to a lack of targeted remodeling typically initiated by the now deceased osteocytes (Schaffler et al. 2014; Burr, 2011; Stout, Cole and Agnew 2019). Osteocyte apoptosis can also occur as a result of limited or complete mechanical disuse (Aguirre et al. 2006). As empty osteocyte lacunae become mineralized through osteopetrosis and proinflammatory cytokines initiate uncoupled osteoclast activity, the bone will become more porous (Herman et al. 2010).

Extensive fracture and age are commonly observed as a contributor to osteocyte cell death, but apoptosis can also occur in the absence of microdamage and in younger individuals through hormone irregularity, mechanical disuse, or under pathological conditions (Komori 2012; Allen and Burr 2014a). Since the potential for osteocytes to regulate metabolic processes depends on their ability to communicate throughout the regional bone environment, pathological conditions that affect the deposition of osteoid, such as Vitamin C deficiency in scurvy, could interrupt the osteocyte communication network and disrupt the coupling effect of bone remodeling that is essential for bone maintenance (Brickley et al. 2020). In another pathological circumstance, studies of people with bone lesions due to multiple myeloma have shown that osteocyte productivity is unregulated, inhibiting cell communication for bone formation. At the same time, osteoclast formation is stimulated due to increased osteoclastogenesis resulting in increased bone porosity (Komori 2012; Delgado-Calle, Bellido, and Roodman 2014). Effectively, osteocytes are the guardians of our skeleton's bone quality and protect it from failure by regulating its mechanical adaptation, mineral homeostasis, and metabolic processes (Hunter and Agnew 2016).

2.2.4. BONE LINING CELLS

Bone lining cells are specialized cells that appear in the reversal phase of bone remodeling to line the surface of the cutting cone after bone has been resorbed (Miller and Jee 1987). Everts and colleagues found that bone-lining cells in the long bones and calvaria of mice remove the remaining collagen fibers exposed by osteoclast resorption and deposit a thin, wavy, mineral-deficient, and sulfur-rich collagenous matrix that demarcates the osteon boundary creating what is known as the cement or reversal line (Everts et al. 2002). The cement line is composed of proteoglycans like osteopontin (Robling, Castillo, and Turner 2006) and while it is not entirely agreed upon whether the cement line is hyper or hypo-mineralized, it is becoming clearer that bone-lining cells function as an important contributor to the successful remodeling of bone (Everts et al. 2002).

2.3. SKELETAL GROWTH AND DEVELOPMENT

While the rate and pattern of human bone growth and development varies depending on skeletal element, age, sex, nutrition status, lifestyle, and genetics (Armelagos et al. 1972; Carter, Van der Meulen, and Beaupré 1996; Wescott 2006; Floyd 2007; Kuzawa 2007), there are only two

modes by which ossification occurs in vertebrates: intramembranous ossification and endochondral ossification. This section briefly describes these two processes and the patterns of modeling and remodeling that build and maintain bone structure as a basis for understanding the function of bone histology in bone growth and development. Comprehension of the regular habits of bone is essential for understanding how nutritional deficiencies and diseases such as those experienced by people who lived during the Great Famine can affect these processes.

2.3.1. INTRAMEMBRANOUS OSSIFICATION

Intramembranous (or desmal) ossification occurs without a cartilaginous model and forms the flat bones that make up the skull as well as the scapula and the pelvis (Martin et al. 2015). Embryonically, this development process begins to produce woven bone, the immature, unorganized, and quickly formed bone structure, within a primitive connective tissue called mesenchyme. When mesenchymal stem cells consolidate, they form what is referred to as a bone blastema before the transcription factor RUNX2 differentiates these cells into osteoblasts to excrete the bone building matrix osteoid. The initial deposition of osteoid produces a primary ossification center, the origin of bone formation. Here, the bone begins the process of resorption and formation in response to genetic predisposition and mechanical demand to create longitudinal growth (Maggiano 2012).

2.3.2. ENDOCHONDRAL OSSIFICATION

Endochondral ossification is the process by which all other bones of the skeleton are formed. It is different from intramembranous ossification in that prior to bone formation, cartilage building cells called chondroblasts are differentiated from mesenchymal cells through transcription factor SOX-9. This creates a hyaline cartilage template for mineralized bone tissue to form. Similar to osteoblasts, chondroblasts secrete a cartilage building matrix that can sometimes incorporate them to create chondrocytes (Allen and Burr 2014).

The cartilaginous model of the bone-to-be is surrounded by a membranous perichondrium that initiates growth through surface cells that later differentiate into osteoblasts through transcription factor RUNX2. Then, osteoblasts deposit their bone building matrix along the surface of the cartilaginous template to create a ‘bone collar’ of primary lamellar bone. In long bones the bone collar first forms along the diaphysis of a developing element, transitioning the

surface from perichondrium to periosteum. The bone collar functionally restricts nutrients to the internal chondrocytes causing them to die and become mineralized. A marrow cavity is formed when primary vessels infiltrate the bone collar and provide nutrients to osteoblasts while delivering osteoclasts that remove the hypertrophied chondrocytes. At this time, primary ossification centers are formed while secondary ossification centers in the epiphyses of long bones develop a little later (Allen and Burr 2014).

2.3.3. MODELING

Skeletal modeling is a subprocess of growth that occurs through the independent efforts of bone resorption and deposition at both the periosteal and endosteal membranes (PEM), to dictate the geometry of bone during growth and development (Maggiano 2012; Martin et al. 2015). While the ossification process causes bones to grow in length, the goal of modeling is to shape the bone while also increasing bone size and developing the bone's shape for optimal functional morphology (Maggiano 2012; Allen and Burr 2014; Martin et al. 2015). Modeling at the PEM is a diametric process of resorption and appositional bone formation that guides the bone's curvature during what is referred to as "bone drift" (Agnew et al. 2013; Maggiano et al. 2016). Bone drift is the change of the position of the cortex of the bone relative to its central axis, causing the bone to alter in size and shape for adaptive purposes (Allen and Burr 2014; Agnew et al. 2013) through the resorption of bone on one side and the deposition of bone on the opposing side (Figure 2.2.). The collaborative action of bone precursor cells, osteoblasts, and osteoclasts is necessary to normalize strains throughout the cortex and create the shape of the bone, a feature that is specific from person to person and across populations (Martin et al. 2015). When tissue is strained through biomechanical loading, for example, modeling is signaled to initiate in two stages: 1) activation by recruitment of precursor cells, and 2) either formation or resorption. New bone is necessary when strain thresholds are exceeded, while low strains call for resorption of bone at that location.

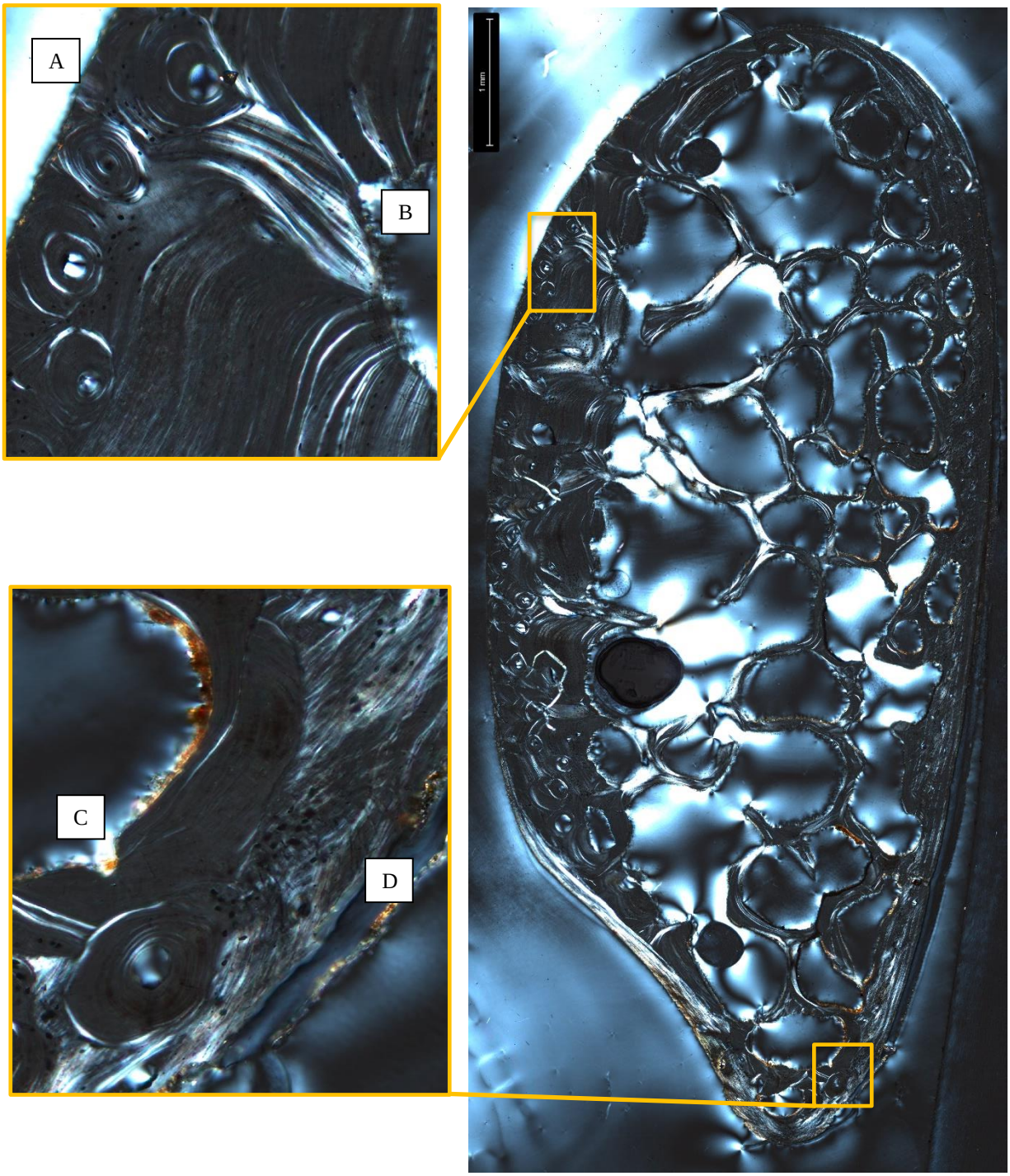


Figure 2.2. Remodeling drift in a young child (DXCIII) showing secondary osteons along the periosteum (A) and unremodeled lamellar bone on the endosteal aspect (B) of the pleural cortex and secondary osteons on the endosteal border (C) with unremodeled lamellar bone along the periosteal border (D) of the cutaneous cortex (10x magnification under polarized light; scale set at 1 mm).

While remodeling is a lifelong process, modeling starts embryonically and continues throughout growth and development. It slows as bone matures (Stout and Crowder 2012; Martin et al. 2015); however, it can occur in older age during fracture repair, loading of pathological bone, and at the postmenopausal period in women when periosteal modeling is stimulated by the decrease in estrogen hormone and an imbalance of bone remodeling occurs (Peck and Stout 2009).

2.3.4. REMODELING

Throughout life, bone is maintained and repaired by remodeling, the coupled action of osteoblasts and osteoclasts within a structure called the basic multicellular unit (BMU) (Martin et al. 2015). While *modeling* is described as formation and resorption occurring along opposing endosteal and periosteal bone surfaces, cortical bone *remodeling* involves formative and resorptive processes occurring on the same envelope (i.e., endocortical, periosteal, trabecular, and intracortical) at the same time. Remodeling can take place at the same time modeling occurs throughout the cortex as both serve different functions over time. However, while modeling relies on the independent actions of osteoclasts and osteoblasts, remodeling of bone tissue occurs in five primary coordinated stages: activation, resorption, reversal, formation, and resting (Frost 1969; Henriksen et al. 2009) (Figure 2.3).

The first stage, *activation*, refers to signals transferred by osteocytes that trigger precursor cell differentiation into osteoclasts. Then, osteoclasts begin removing bone from a particular site that is needed to repair or simply maintain cortical bone density and strength. The activation signals are transmitted in response to microdamage and osteocyte apoptosis in targeted remodeling and/or non-location specific stochastic remodeling, a method of maintaining mineral homeostasis (Martin 2002; Eriksen 2010). The activation rate of remodeling in juvenile bone during growth; however, is high relative to adults due to the rapid rate of turnover necessary for development. While this does lead to increased resorption and high porosity, this porosity has been observed in normal subadult bone and may not actually be indicative of a metabolic issue (Sedlin, Villanueva, and Frost 1963).

After osteocytes activate a remodeling effect, the second stage, *resorption*, begins as osteoclasts within the BMU remove the necessary bone creating a tunnel, or ‘cutting cone’, along the bone

surface or within the cortex (Figure 2.4). Osteoclasts move at about 40μm/day longitudinally and 5μm/day radially and tunnels are typically 200μm in length. In a transverse cross-section, the cutting cone feature is observed as a resorption bay and can reach a diameter of 150-350μm over the course of three weeks in adults (van Oers et al. 2008). Resorption bays are the features that show scalloped edges when osteoclasts and are resorbing the bone and, combined with Haversian canals, make up the portion of the cortex referred to as porosity area and percent porosity in this thesis. A stage called *reversal* is often referred to as the change from osteoclast resorption to bone formation by osteoblasts either by cell-to-cell communication or through the release of growth factors during resorption (Allen and Burr 2014). This stage also involves the clearing of the remaining bone in the resorption pit and deposition of the outer boundary of the osteon by bone lining cells to form the cement line (Henricksen et al. 2009; Allen and Burr 2014).

In the next stage known as *formation*, a bone structural unit (BSU) is created, referred to in bone biology as a Haversian system or an osteon. As the cutting cone burrows through the cortices to remove old primary or secondary bone, osteoblasts replace the resorbed bone with osteoid. While osteoblasts deposit osteoid and fill in the tunnel they leave room for a central canal, referred to as a Haversian canal, to provide nutrients including calcium and phosphate throughout the bone. Nerve and blood supply are also dispersed through the bone via Haversian canals (Martin et al. 2015). At the same time, bone-lining cells blanket the cutting cone, removing leftover collagen fragments and sealing the osteon in a hyper-mineralized reversal line, described earlier as a cement line (Parfitt 1984; 2001). The formation process takes about three months to complete and is followed by a period of collagen fiber mineralization and primary mineralization of osteoid. Quiescence, or the *resting* phase, is considered the final phase wherein bone lining cells continue to mineralize the bone surface. All in all, about 3% of adult cortical bone is replaced each year and each full process of remodeling takes four to six months to complete depending on age and the metabolic and mechanical integrity of the bone and body (Allen and Burr 2014; Martin et al. 2015).

The remodeling process is a balancing act, monitored by delicate sensors that dictate how bone adjusts for the mechanical loading environment and nutritional demand. However, nutritional deficiencies or age-related processes that deplete calcium reserves in the skeleton can disrupt

the balance or remodeling creating low-density porotic bone that is vulnerable under mechanical demand (Agnew and Stout 2012). In cases that cause osteoporosis, activation and resorption are acting at a rate greater than formation. In other words, there is a lag in the time between bone being taken away and mineralized bone being deposited, leaving a skeleton that is more likely to fracture. This type of disruption in remodeling occurred in monkeys (*Cercopithecus griscozliridis* and *Cercopithecus arthiops*) who developed scurvy after eighteen weeks of being fed a maize based diet. In this study the monkeys experienced long bone growth arrest, loosening of teeth, bleeding gums, and cross-sections of the long bones revealed failure of secondary osteon replacement of primary bone, resulting in more immature bone histology than expected for age (Follis 1957).

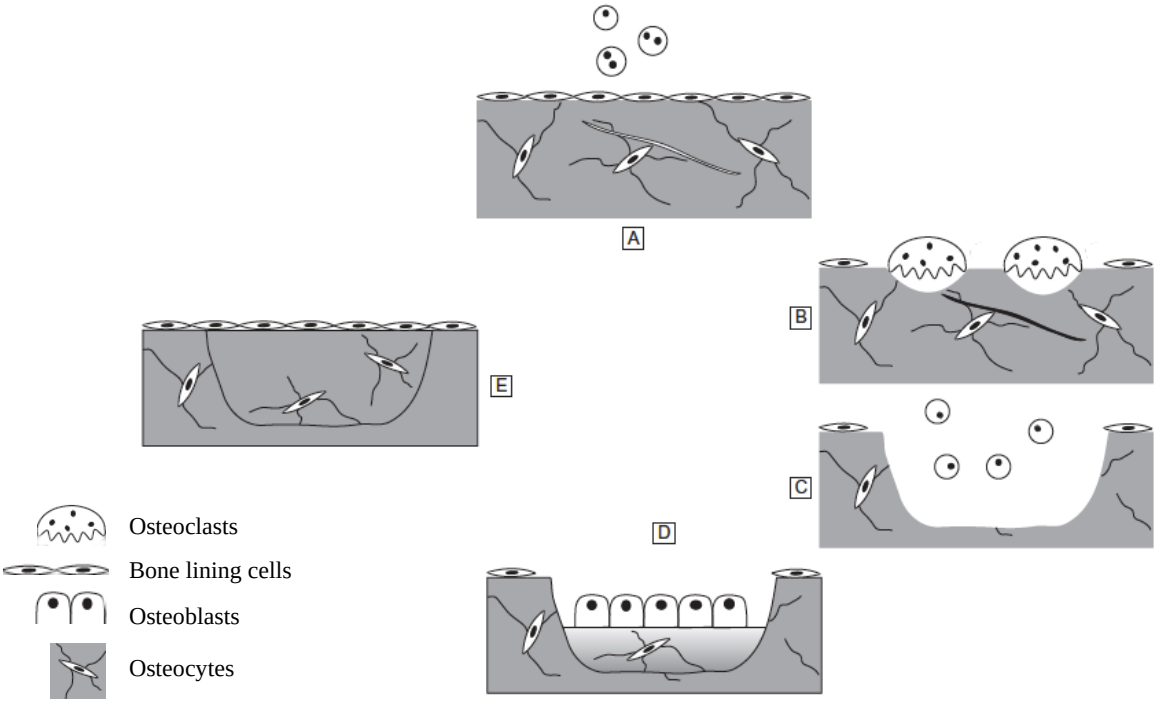


Figure 2.3. The stages of remodeling in fracture repair: A) Activation; B) Resorption; C) Reversal; D) Formation; E) Resting. Source: Adapted from Allen and Burr 2014

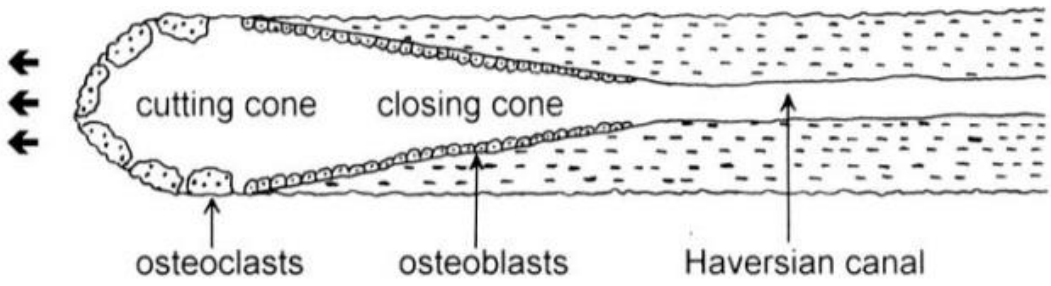


Figure 2.4. Illustration of a cutting cone showing osteoclasts resorbing bone while osteoblasts refill the cortex surrounding a central Haversian canal. Source: Adapted from Chamberlain and Forbes 2005

2.4. HUMAN BONE TYPES AND HISTOLOGICAL STRUCTURES

There are three main types of human cortical bone that can be recognized under the microscope for histological investigations: woven, lamellar, and secondary lamellar bone. Each type indicates a different stage of growth and development while variations within the type of bone can provide information about behavior, diet, and disease. This section describes the differences between cortical bone and trabecular bone, each cortical bone type and how it can be identified, and the histological structures within these cortical bone types. Emphasis is placed on histological structures because they are an integral part of the bone remodeling process and are used to infer the health status of the Kilkenny Union Workhouse population sample studied in this thesis.

2.4.1. WOVEN BONE

Woven bone is the immature primary or fetal bone. It is named for its mesh composition of loosely knit basket-like collagen fibers. Under polarized light woven bone is unorganized and hyper-mineralized (Streeter 2010) (Figure 2.5). This type of bone is formed from osteoblast secreted osteoid in utero and as bone mineralizes in infancy and childhood. Woven bone is laid down quickly to form a basis for remodeling into more structurally sound lamellar bone (Streeter 2010). While it is commonly associated with bone modeling in childhood, woven bone also appears throughout life as the first type of bone to form in the process of healing fractures or at pathological lesion sites (Buckwalter et al. 1995; Athanasou 2009). For example, Ortner and Mays found patchy deposits of woven bone in the ribs and crania of children exhibiting Vitamin D deficiency (Ortner and Mays 1998) and von Hunnius and colleagues, found evidence of woven bone in the tibia of a 25-39 year old adult with macroscopic evidence of syphilis (Hunnius et al. 2006).

2.4.2. PRIMARY LAMELLAR BONE

When woven bone is remodeled by the coupled action of osteoclasts and osteoblasts it becomes primary, or unremodeled, lamellar bone, also known as circumferential or interstitial lamellar bone (Streeter 2010; Stout et al. 2019). Primary lamellar bone is the intermediate bone type recognized three-dimensionally by its plated structure wherein sheets of bone overlap to form a strong foundation of mature, tightly packed, collagen fibers (Streeter 2010). Under polarized

light in a transverse cross-section of bone, this type of lamellar bone is identified by streaks of bright, wavy lines punctuated by primary vascular canals or between secondary osteons (Figure 2.5). These canals, known as primary osteons, are blood vessels that became trapped in the compact bone during radial growth (Crowder and Stout 2011; Stout et al. 2019).

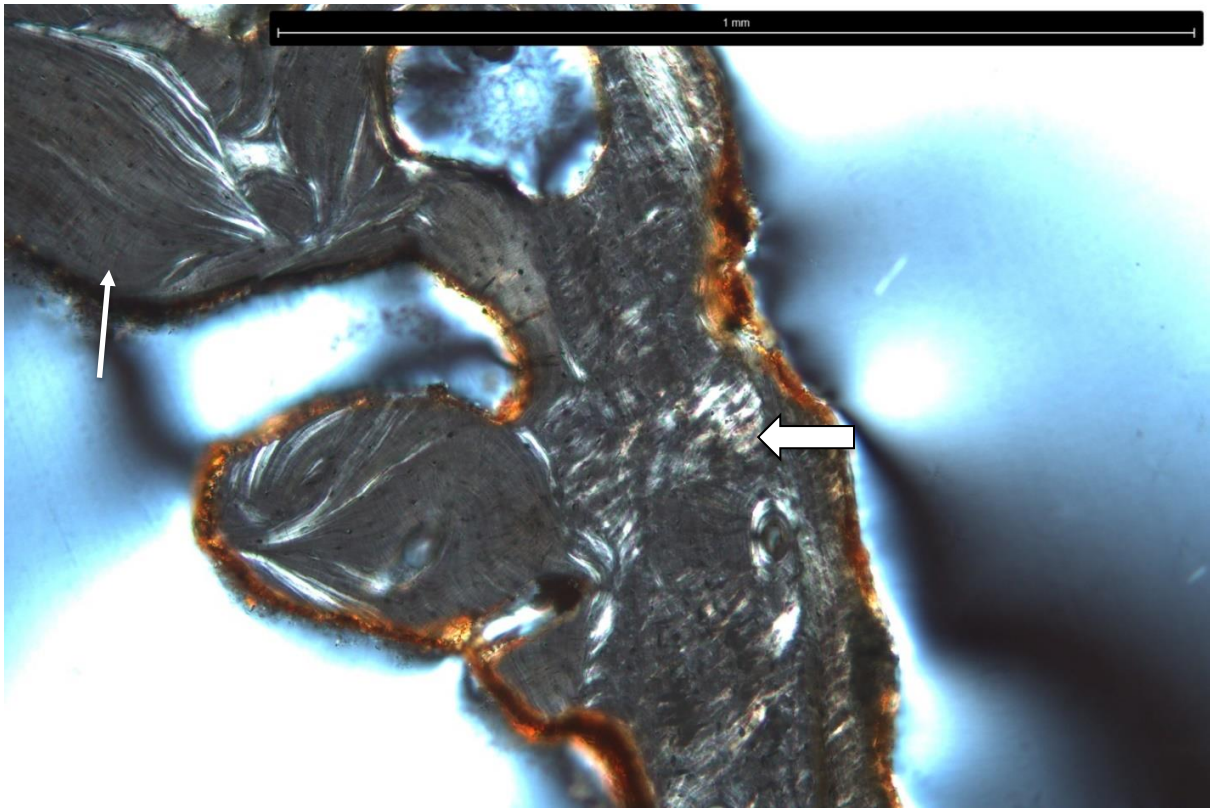


Figure 2.5. Woven bone (thick white arrow) and primary lamellar bone (thin white arrow) in the middle adult rib of a young child (CCXCVI) estimated to be about 2.5 years old (10x magnification under polarized light; scale set at 1 mm)

2.4.3. SECONDARY LAMELLAR BONE

As primary lamellar bone is further remodeled, it becomes populated by secondary lamellar or osteonal bone, also known as concentric lamellar bone (Figure 2.6). This terminal type of bone, named and identified by secondary remodeling events called osteons, is the strongest of the three types of bone (Streeter 2010). The tightly bound collagen fibers and cylindrical form of secondary lamellar bone allow it to resist compression and slow the propagation of fractures (Stout and Crowder 2012). As osteons continue to fill the cortex of the bone, older osteons from earlier remodeling events are removed by osteoclasts to make room for new osteons.

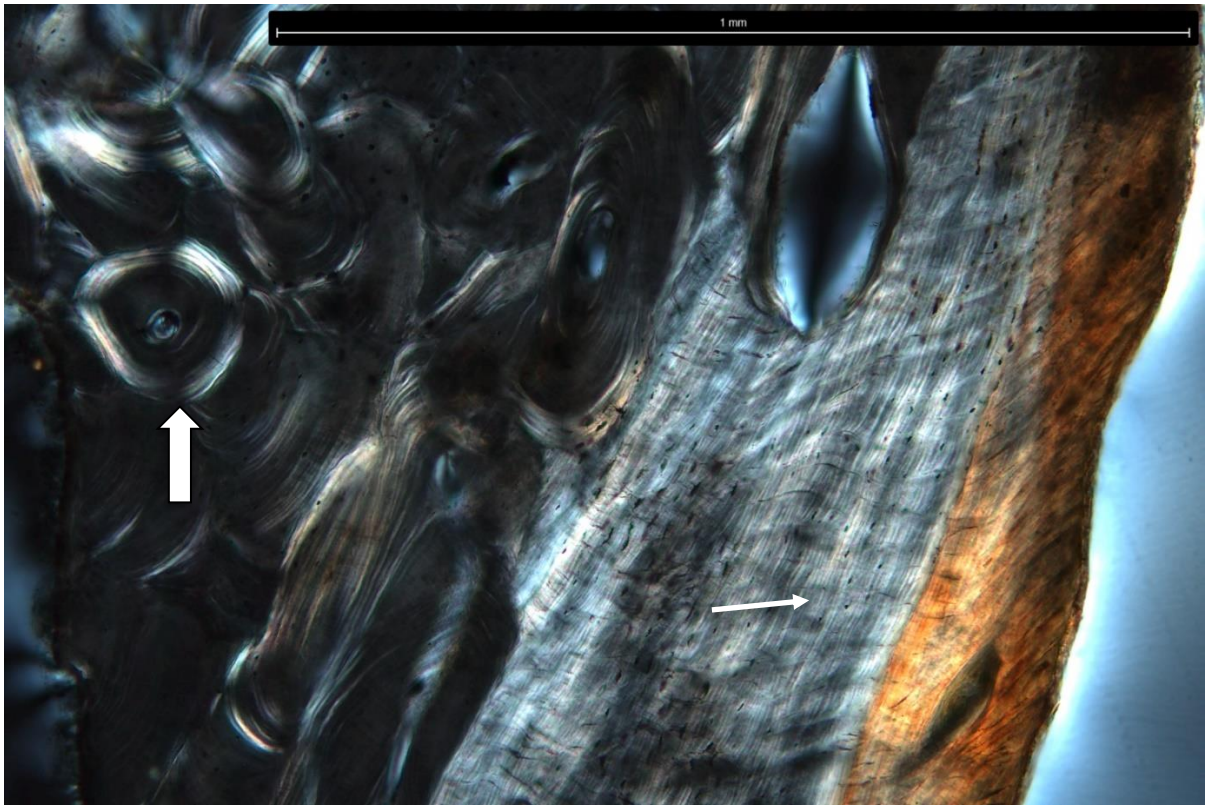


Figure 2.6. Secondary lamellar bone (thick white arrow) and primary lamellar bone (thin white arrow) in the middle rib of an adolescent (CLIX) estimated to be about 13 years old (10x magnification under polarized light; scale set at 1 mm)

2.4.4. OSTEONS

As mentioned above, osteons are the secondary remodeling events that populate cortical bone and make up the third stage of bone remodeling referred to as secondary lamellar bone. There are a few different types of osteons that can be observed under bright or polarized light and result from variations remodeling patterns.

Type I or "intact" osteons are the result of the cutting cone, the goal of burrowing osteoclasts and osteoid depositing osteoblasts. When viewed in transverse cross-section, Type I osteons are circular structures with a bright outer reversal line and multiple layers of collagen (concentric lamellae) (Figure 2.7). They contain calcium phosphate crystals to help mitigate microfracture propagation and keep bone strong when compression forces act upon it (Martin 1991). These concentric collagen and mineral layers surround and protect a central channel called a Haversian canal that distributes blood, nerves, and nutrients throughout the bone (Raguin and Streeter 2018). Longitudinally, Type I osteons are cylindrical, tube-like structures

that fill the cortices, but they change in quantity, size, and shape with remodeling over time (Hennig et al. 2015). Many archaeological and forensic studies have used osteon size to aid in age estimation (Cho and Stout 2003; Burr, Ruff, and Thompson 1990; Stout and Paine 1992), to separate human from non-human bone (Crescimanno and Stout 2012; Tersigni 2005), and to identify differences due to mechanical loading environment (Miszkiewicz 2016). However, Pfeiffer and colleagues did not find any correlation between osteon size and biomechanical or metabolic activity (Pfeiffer et al. 2006).

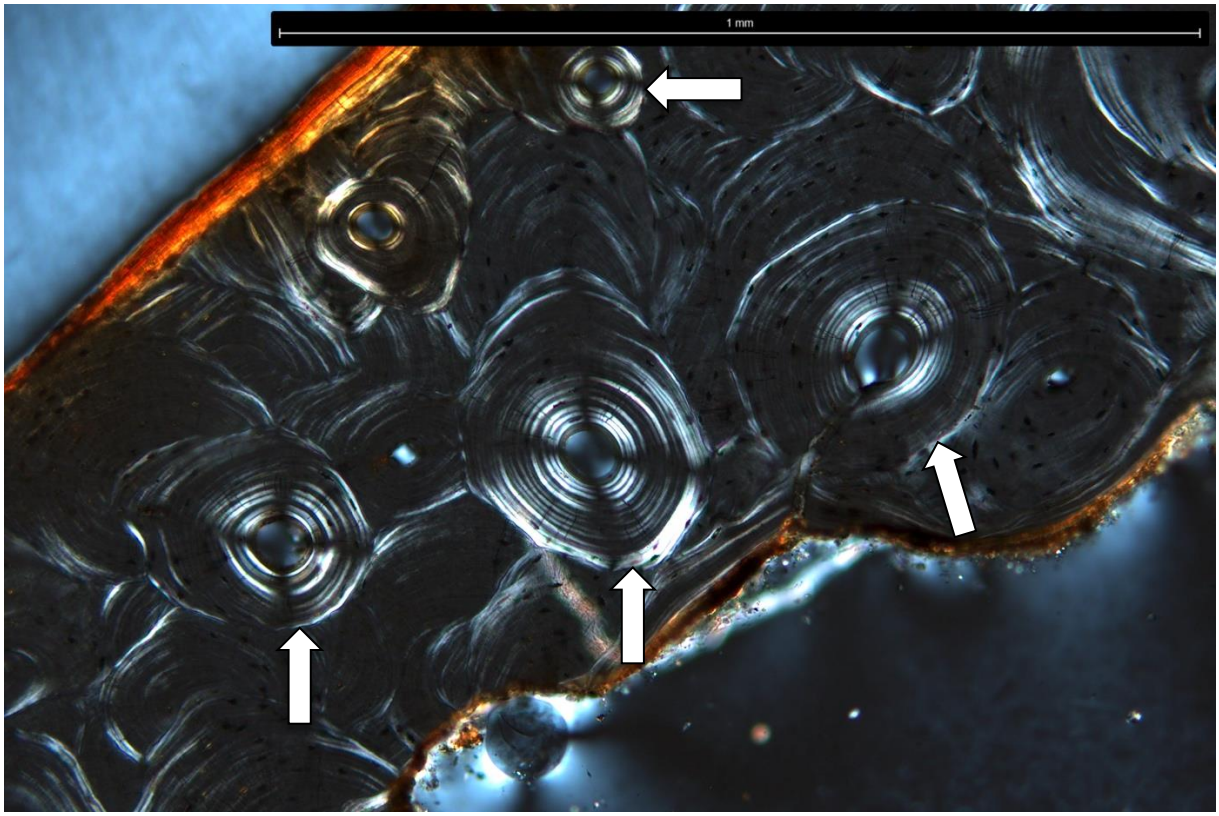


Figure 2.7. Type I osteons in the middle rib of an older adult male (CDLXXXVI) with a point age estimate of 50.6 years (10x magnification under polarized light; scale set at 1 mm)

Type II osteons, also known as embedded osteons, are intact osteons that have remodeled inside an existing osteon, filling in the existing Haversian canal and leaving evidence of the previous osteon only by the presence of a bright outer concentric ring (Figure 2.8). Since the concentric lamellae of the two osteons do not align, the two osteons are distinguishable from one another—one new, embedded osteon and one that has become an osteon fragment as a result of the remodeling process (Stout et al. 2019). Type II osteons are thought to be linked to age and potentially associated with dietary changes (Ortner 1975; Yoshino et al. 1994).

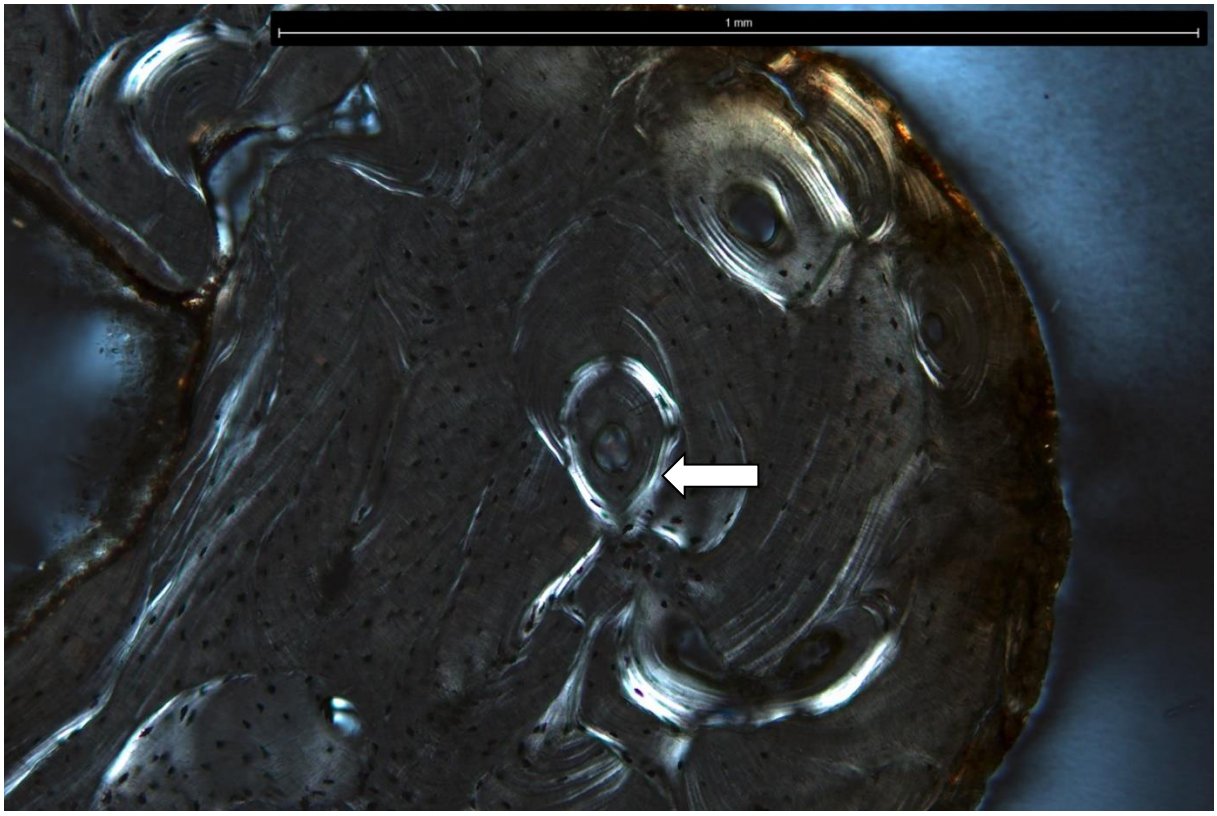


Figure 2.8. Type II osteon in the middle rib of an early middle adult female (CCLXXXIX) with a point age estimate of 29 years old (10x magnification under polarized light; scale set at 1 mm)

Osteon fragments, a third osteon type, are the remnants of older Type I or Type II osteons that have been partially remodeled by newer osteons and are still identifiable by the presence of a portion of their outer reversal line (Stout and Paine 1992) (Figure 2.9). Changes in osteon morphology from secondary (*intact* osteons) to osteon fragments occur in a somewhat predictable pattern as new osteons continue to remodel over older osteons creating more fragments over time. A predictable pattern of remodeling is useful for bone histological analysis of archaeological samples because it allows researchers to infer aspects of life history such as the stage of life the person was in when they died and the determine if stress factors like diet or disease have impacted the remodeling process (Hennig et al. 2015; Dominguez and Agnew 2016).

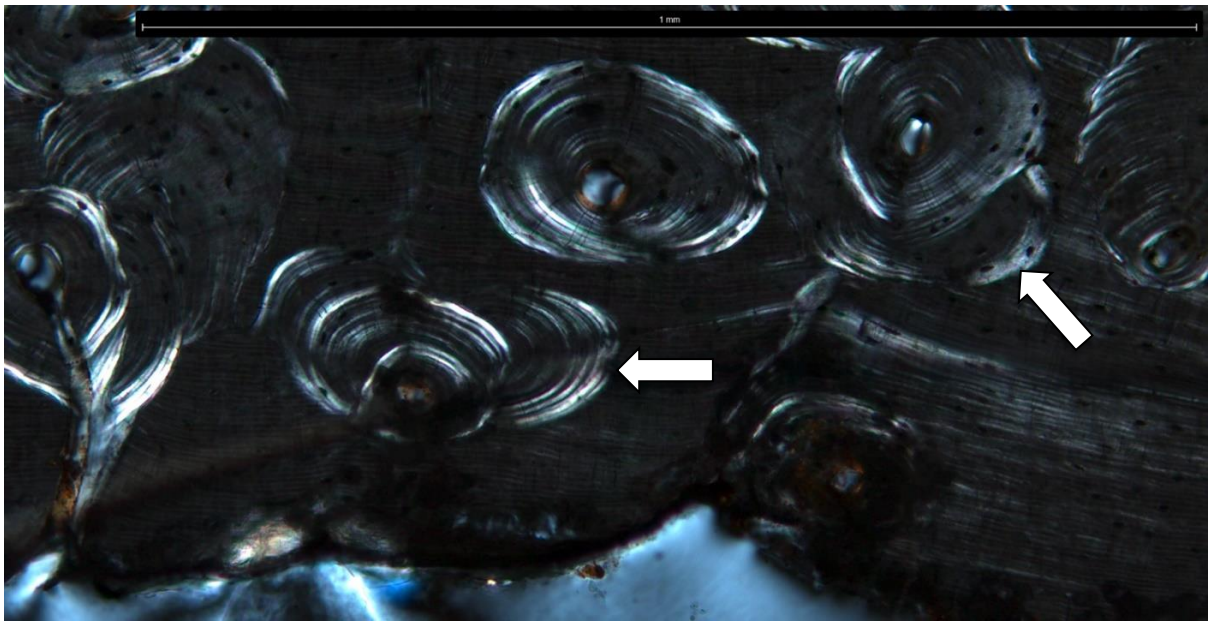


Figure 2.9. Osteon fragments in the middle rib of a late middle adult female (CXXXIII) with a point age estimate of 40.6 years old (10x magnification under polarized light; scale set at 1 mm)

The quantification of the cumulative number of intact and fragmentary osteons within secondary lamellar bone, referred to as osteon population density (OPD), can indicate the activation rate at which bone is remodeling (Wu et al. 1970; Stout and Lueck 1995) and is a primary variable used in histological age estimation methods for modern and archaeological populations (Thompson 1979; Ericksen 1991; Cho and Stout 2003; Paine and Brenton 2006; Suzuki and Maggiano 2018). In a recent study, Suzuki and Maggiano found that modern Mayan OPD was more similar to prehistoric Mayan OPD than the OPD of a modern Hispanic sample (Suzuki and Maggiano 2018). These results indicate possible biocultural differences in the rate of osteon remodeling in the ribs between the two modern samples (Cho et al. 2003; Suzuki and Maggiano 2018). While this study supported the utility of OPD for age estimation over other variables such as osteon size and shape for predicting age, it also emphasized that biocultural differences should be considered when modern methods are used to create biological profiles for archaeological groups.

Double zonal osteons are another type of osteon found in the transverse cross-section of human bone. Double zonal osteons contain areas of variable mineral density that can be identified by the presence of a hyper-mineralized concentric ring within the Haversian system (Figure 2.10). This line is contained within the osteon itself and should not be confused with the outer reversal

line of the osteon (Pankovich et al. 1974). The significance of double zonal osteons is not well understood (Austin and Mulhern 2015), but studies have shown they are related to changes in collagen orientation and mineralization (Raguin and Streeter 2018), factors that are dependent on osteoblast activity and affect the structural integrity of bone. Since collagen orientation is related to strain influences, Raguin and Drapeau have proposed that double zonal osteons may occur due to variations in mechanical load distribution, however further investigation determined that the relationship between the frequency of double zonal osteons and loading environments is weak and requires further investigation (Raguin and Drapeau 2020).

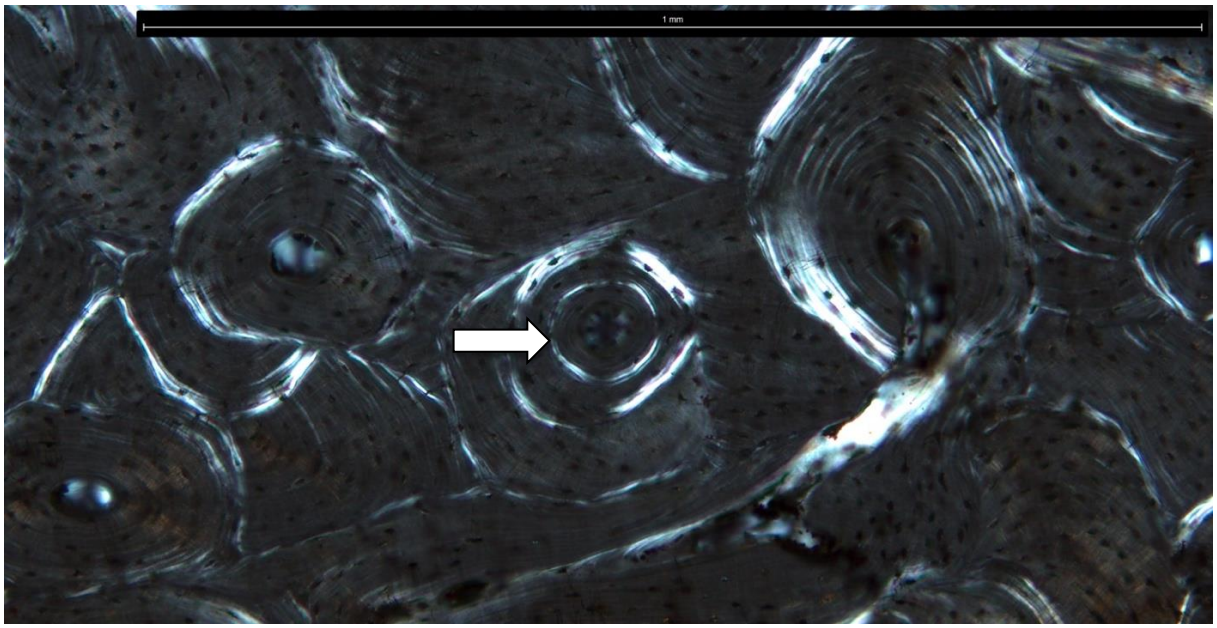


Figure 2.10. Double zonal osteon in the middle rib of an adolescent (DLXXXII) with a point age estimate of 14.5 years old (10x magnification under polarized light; scale set at 1 mm)

The dominant hypothesis on the cause of double zonal osteons has been that this type of osteon represents a disturbance or arrest in the mineralization process due to a period of stress, similar to the proposed etiology of enamel defects or Harris lines (Martin and Armelagos 1985; Austin and Mulhern 2015; Raguin and Streeter 2018; Reichert and Mulhern 2018). This idea is supported by Reichert and Mulhern who found that while there were no significant differences in the overall size of Type I osteons and double zonal osteons, the latter do have smaller Haversian canals than Type I osteons. The authors suggest this finding means fewer nutrients were available during the formation of the double zonal osteon resulting in the development of a functionally smaller canal (Reichert and Mulhern 2018). Additionally, Brickley and

colleagues found “defective” cement lines in a sample of individuals from the eighteenth and nineteenth centuries with osteomalacia (Brickley et al. 2007). Higher frequencies of double zonal osteons were found in ribs with greater cortical area in a sample of archaeological remains from Sudanese Nubia (Martin and Armelagos 1885). The authors suggest the appearance of these osteons indicates recovery from growth arrest that did not occur in individuals with smaller cortices (Martin and Armelagos 1985). The fact that double zonal osteons are frequently found in populations that experienced multiple states of stress, including disease and malnutrition indicates hypermineralization is most likely related to prior episodes of nutrient deficiency.

Another type of osteon known as a drifting osteon is also found in cortical bone but is more prevalent in the cortices of young adults and children (Epker and Frost 1965). These osteons are identified by their extended “tail” created by the continuous resorption and deposition of bone on opposing sides and, like Type I and Type II osteons, are a function of normal histomorphological variation (Epker and Frost 1965; Streeter 2010) (Figure 2.11). Due to their known relationship with age and lack of evidence for a relationship with paleopathology, drifting osteons will not be quantified for this research.

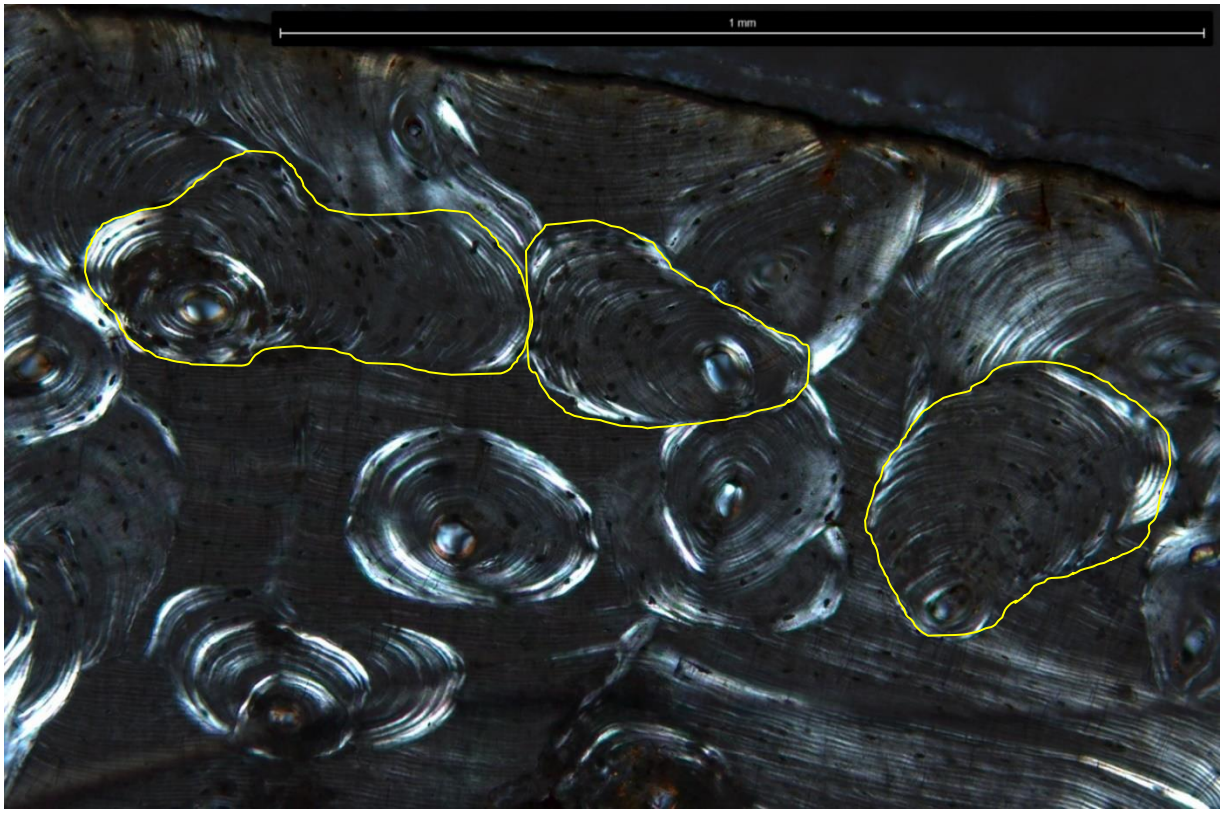


Figure 2.11. Drifting osteons (yellow outline) in the middle rib of a late middle adult female (CXXXIII) with a point age estimate of 40.6 years old (10x magnification under polarized light; scale set at 1 mm)

2.4.5. RESORPTION BAYS

Resorption bays, as described earlier, appear under the microscope in transverse cross-section as large voids with scalloped edges in the cortical bone (Figure 2.12) (Landeros and Frost 1964; Richman, Ortner, and Schulter-Ellis 1979). Resorption bays are the product of BMU resorption in the process of bone remodeling and may be of any shape or size but are larger than a Haversian canal (>40-50 μm in humans) and can potentially dictate the size of the osteon that will form in its place (Sedlin, Villanueva, and Frost 1963). Resorption bays are known to make up the composite of "porosity" within a cortical region (De Boer, Van der Merwe, and Maat 2013). If osteoclastic resorption occurs at a rate greater rate or in the absence of osteoid deposition, it is possible for a number of resorption bays to increase and coalesce, contributing to greater intracortical porosity area and percent porosity within the cortex. The amount of porosity, percent area of porosity, and the percent area of cortical bone can inform researchers on the state of the bone at the time of death, including whether or not the individual was experiencing difficulty in maintaining "normal" remodeling rates and metabolic homeostasis

(De Boer, Van der Merwe, and Maat 2013). For example, osteoporosis is a widely recognized consequence of poor health due to increased cortical porosity that results from the failed or uninitiated refilling of bone excavated by osteoclasts (Agarwal 2012a). While osteoporosis is typically understood as an age-related issue, osteoporosis can occur as a response to disease processes where the homeostatic process of remodeling is disrupted, such as in the increased resorption and lack of osteoid formation in the non-pathognomonic effects of scurvy (Fain 2005; De Boer, Van der Merwe, and Maat 2013).

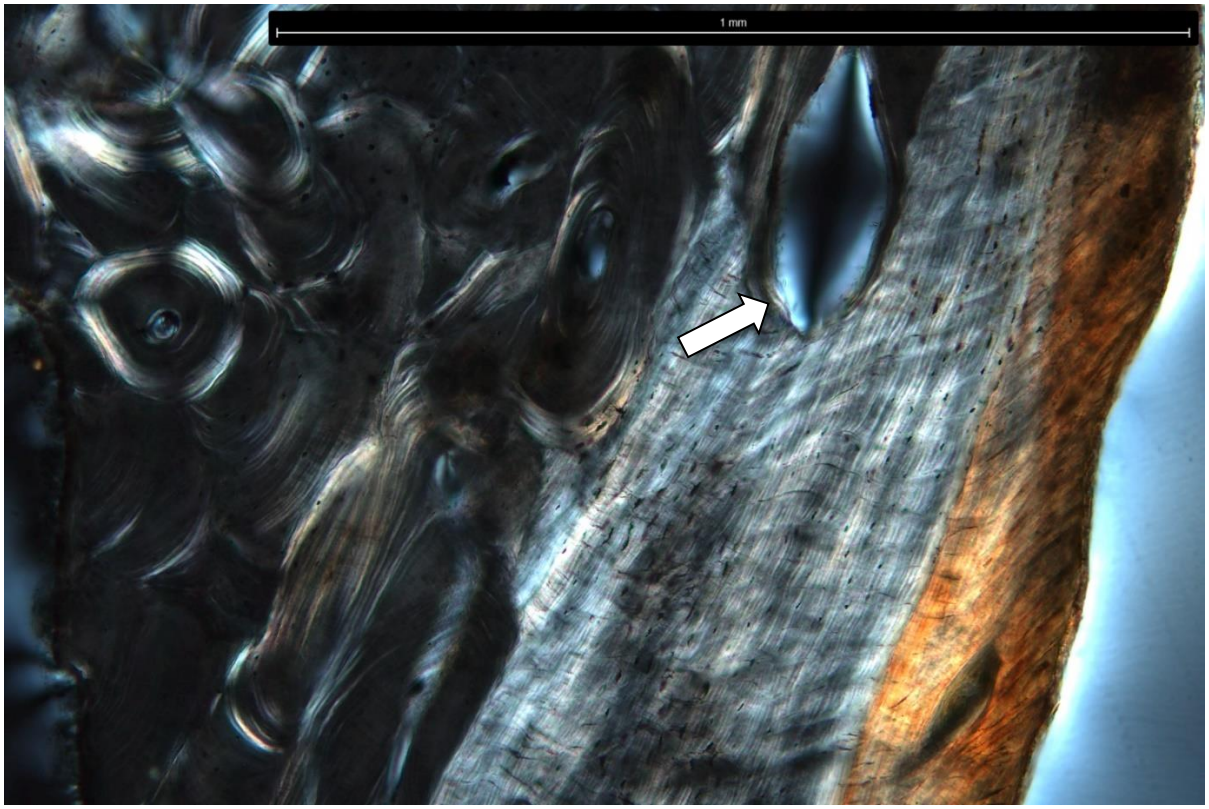


Figure 2.12. Resorption bay in the middle rib of an adolescent (CLIX) estimated to be about 13 years old (10x magnification under polarized light; scale set at 1 mm)

2.4.6. TYPE 1 COLLAGEN

At the nano-level, the skeleton maintains its plasticity and elasticity through its organic (collagen) and inorganic (mineral) components. Bone is partially made up of oxygen and hydrogen, but it is primarily composed of calcium and phosphorus, which it stores and shares with the rest of the body. These mineral platelets are interspersed between anisotropic cross-linked collagen fibers and non-collagenous proteins that lie within the interstitial lamellae of primary and secondary lamellar bone (Ruppel, Miller, and Burr 2008). The orientation of these

collagen fibers and the interspersed mineral component is what determines the mechanical strength (hardness, stiffness, and toughness) of the bone in either direction (Ruppel, Miller, and Burr 2008). For example, cortical bone is best suited to resist compression loads due to the orientation of its collagen fibers (Skedros et al. 2013). Osteons themselves act as longitudinally oriented bundles of fiber and the reversal line is adept at protecting the whole bone by absorbing energy, reducing crack propagation, and controlling fatigue (Martin et al. 2015). Therefore, bone becomes more frail when collagen formation is inhibited through nutritional deficiency (Kipp et al. 1996; Peterkofsky 1991; De Boer, Van der Merwe, and Maat 2013; Brickley, Ives, and Mays 2020b).

2.5. APPLICATION OF BONE HISTOLOGY IN BIOARCHAEOLOGICAL AND PALEOPATHOLOGICAL RESEARCH

Bioarchaeology is an interdisciplinary field of study that incorporates osteology with related fields such as archaeology, pathology, and biochemistry. The discipline contributes to the reconstruction of past human lives by examining skeletons from archaeological sites (Buikstra and Beck 2006). Paleopathology, a sub-field of bioarchaeology, is the study of health and disease experienced by the people discovered at these sites (Ortner 2011). The experience of stress in the past is individualistic and complex but identifying frequencies of pathological changes in skeletal samples can help researchers reconstruct social dynamics and demographic patterns that contribute to the understanding of health and susceptibility of disease for archaeological populations. Evidence of physiological stress in the skeleton can be detected through growth disruptions such as enamel defects, Harris lines, stunted stature, or through the interpretation of osseous lesions—the pathological changes in bone (Goodman 1981; Boldsen, Milner, and Weise 2015; Larsen 2018; Reichert and Mulhern 2018; Brickley, Ives, and Mays 2020a). These lesions may be remnants of non-specific, degenerative, metabolic, or infectious disease and in some cases the disease may be specifically diagnosed.

Bone histology is a multi-functional technique that has been used to analyze archaeological and modern human skeletal samples for many purposes, including the determination of human from non-human bone (Mulhern and Ubelaker 2012; Crowder, Andronowski, and Dominguez 2018), exploration of the impact of behavior and subsistence patterns within and between populations (Burr, Ruff, and Thompson 1990; Stout and Lueck 1995; Pfeiffer et al. 2006), to estimate age

(Stout and Paine 1992; Cho and Stout 2003; Brenton and Paine 2006; Pfeiffer et al. 2016; Suzuki and Maggiano 2018), and to infer population health and diet (Wu et al. 1970; Stout and Teitelbaum 1976; Richman, Ortner, and Schulter-Ellis 1979; Martin, Magennis, and Rose 1987; Peterson and Riggs 2010). As discussed in Section 2.4., bone histological variables, while initially determined by genetic heritability, can adapt in size, shape, and quantity primarily to satisfy various mechanical and metabolic demands throughout life (Bjørnerem et al. 2015; Agarwal and Miller 2016; Eleazer and Jankauskas 2016; Beresheim, Pfeiffer, and Alblas 2018). These adaptations help bone tissue reach optimal strength and toughness to resist fracture. However, similar to the macroscopic features of bone observed in paleopathological analyses, histomorphology is also influenced by a number of factors including lifestyle, age, social structure, lifestyle, and disease (Burr, Ruff, and Thompson 1990). These biological, environmental, and cultural pressures can cause bone to react systemically throughout the skeleton at the microscale prior to their effect on the macroscopic size and shape of skeletal elements (Kerley 1965). The following sections provide examples for how bone histology can inform researchers about biological and cultural factors and how the method has been used in bioarchaeology to explore these aspects in past populations.

2.5.1. BIOMECHANICS AND BONE HISTOLOGY

The initiation of bone cell activity in response to a change in mechanical loading environments is known as bone functional adaptation (Pivonka 2018). Bone functional adaptation is predicated on the mechanostat, a theoretical model developed by Frost that describes how bone and the surrounding musculoskeletal tissue adapt to local loading environments to maintain optimum strength required to resist tissue failure (i.e., bone fracture) (Frost 1987). While adaptive secular changes occur across populations over time, such as the variation in femur size and shape with the transition to more sedentary lifestyles (Wescott and Zephro 2016), human bone also changes in response to everyday mechanical stimuli (Villotte and Knüsel 2012). These changes are evident in the size and shape of the element, such as in the entheses of limb bones where habitual loading has caused a thickening in the muscle attachment sites of people who lift weights or participate in strenuous agricultural labor (Stout, Cole, and Agnew 2019; Villotte and Knüsel 2013). However, these visible changes are first initiated below the surface of bone where histological properties work to maintain structural integrity.

As discussed previously, the organization of bone tissue is maintained by osteoclast and osteoblast activity initiated by osteocytes that sense small fractures in the bone and signal repair in the local region. This leads to the reconstruction of bone architecture, an increase in bone density, and alteration of mechanical properties (Robling, Castillo, and Turner 2006). It also results in increased rates of remodeling which, under ideal conditions where nutritional needs are met, leads to improved bone strength and resistance against bone loss later in life (Miszkiewicz 2015; Robling, Castillo, and Turner 2006).

Many studies have shown a relationship between bone histological variables and biomechanical loading, particularly in the femur. Additionally, it has been hypothesized that strain magnitude and mode play a large role in the osteon size wherein the compression and tensile pressures placed on weight bearing bones initiates remodeling and influences osteon size (Skedros et al. 1994; Van Oers et al. 2008). For example, Gleiber found that the absence of mechanical stimuli can affect cortical bone maintenance leading to great bone loss over time. In their study on modern individuals, those who were mobility impaired exhibited decreased cortical bone area, increased medullary area, and increased cortical porosity in the femur indicating increased resorption and lack of bone formation in those who were less mobile (Gleiber 2017). Similarly, Schlecht and colleagues observed larger osteons in quadriplegic individuals than in the associated age-matched sample with mobility (Schlecht et al. 2012). Since osteon area has been shown to decrease with remodeling over time (Takahashi, Epker, and Frost 1965), both observations indicate a lack of osteoblastic remodeling with decreased mobility.

The correlation between bone histology and biomechanical loading has also been used to evaluate the effects of social inequality on bone health. Miszkiewicz and Mahoney observed differences in the femoral microstructure between two groups of different socio-economic status in medieval England where those of the higher status had a greater number of osteons as well as larger osteons and Haversian canals than those of the lower class (Miszkiewicz and Mahoney 2016). This study found differences related to mechanical loading and occupational adaptation due to inequality and showed that histological features in the human femur can shed light on the biological effect of socio-economic inequality if factors such as age and sex are accounted for while historical context is considered.

As exhibited by the studies mentioned above, the femur and other weight bearing bones can provide a wealth of information about the impact of mechanical stimuli on bone histomorphometry and contribute to the reconstruction of lifestyles influenced by society and culture in people from the past. However, these patterns of mechanically induced histological variation in the femur and other long bones creates a bias that makes it difficult to control for variation due to disease or other metabolically induced changes in the bone histology. In other words, patterns of modeling and remodeling in bone microstructure vary depending on the element under analysis (Britz et al. 2009). For example, body weight has been observed as a significant biomechanical stimulus acting on the material properties of the femur (Britz et al. 2009) but not the rib (Tommerup et al. 1993; Beresheim, Pfeiffer, and Alblas 2018).

Similarly, Mulhern found sex differences between male and female femora that were not reflected in the rib (Mulhern 2000). Their research showed that females had fewer and larger secondary osteons than males in the femur, indicating potential differences in mechanical loading environments for the sexes in the Kulubnarti population (Mulhern 2000). However, Pfeiffer and colleagues did not find any significant differences for rib osteon area or Haversian canal area for sex or age (Pfeiffer et al. 2006). While osteon size in the femur regularly correlates to loading and other biomechanical factors (Britz et al. 2009; Burr 1992; Schlecht et al. 2012; van Oers et al. 2008), the rib experiences more regularity in strain as respiration is known to be similar across groups, and variation in osteon size and shape appear to change in a predictable pattern over time (Agnew and Stout 2012; Dominguez and Agnew 2016; Goliath, Stewart, and Stout 2016).

2.5.2. HISTOLOGICAL ESTIMATION OF AGE-AT-DEATH FROM THE RIB

As discussed above, physical activity is important for maintaining bone metabolism because biomechanical stimuli promote bone turnover for maintenance of a healthy and strong skeleton. However, age associated patterns in bone remodeling (OPD, cortical area, osteon size and shape) have also been observed. These variables are often used to estimate age and have been found to be consistent within and between populations depending on the element under analysis (Ahlqvist and Damsten 1969; Goliath, Stewart, and Stout 2016; Kerley 1965; Stout and Paine 1992; Suzuki and Maggiano 2018; Thompson 1979).

In bioarchaeology, estimating age-at-death from the skeleton is one of the first components in creating a biological profile, the method of estimating information such as age, sex, stature, and ancestry, which is important for reconstructing paleodemography, understanding risk for morbidity and mortality, and developing hypotheses about the lived experiences of the humans being studied (Sofaer 2006). However, there are many factors that influence the age of the skeleton at the time of death including lifestyle, genetics, biology, and cultural histories that may require population specific age estimation methods (Agarwal 2012b; Bocquet-Appel and Masset 1982; Clark, Simon, and Hubbe 2020; Pratte and Pfeiffer 1999). Although this thesis is not focused on age estimation, understanding the effects diet and disease have on bone remodeling is important for researchers considering techniques for age estimation in archaeological populations that may have experienced sociocultural, environmental, or economic circumstances that led to resource scarcity and disease.

In bioarchaeological analysis, age is typically estimated using gross macroscopic methods that have been developed from populations with known age at death. For example, one commonly practiced method of adult skeletal age estimation is based on the identification of stages that occur in the degeneration of the pubic symphysis of the os coxae (Brooks and Suchey 1990), another is to examine the sternal ends of ribs (İşcan, Loth, and Wright 1984). In children, age can be estimated by measuring long bones, observing epiphyseal ossification, or by examination of dental mineralization and eruption (Ubelaker and Longeway 2019).

Although macroscopic age-estimation techniques are regularly used and improved upon (Boldsen, Milner, and Weise 2015; Clark, Simon, and Hubbe 2020), methods for age estimation based on bone histological analysis are given less attention (Crowder 2009). This is partially due to the destructive analysis of bone histology and the time and equipment necessary to perform the methods, but also because the relationship between bone histology and age is multifaceted and complex (Crowder 2009). On one hand, many studies have shown that there is a correlation between chronological age and certain bone histological variables, notably OPD—specifically in the rib and in the femur but also in the humerus, scapula, tibia, and in some of these elements combined (Cho and Stout 2003; Mulhern 2000; Streeter 2010; Stout and Paine 1992; Thompson and Galvin 1983). On the other hand, chronological age and

biological age of bone do not exist as parallel entities (Agarwal 2012; Halcrow and Tayles 2008). While chronological age is a constant determined by calendar months or years, biological age, or the physical aging of the body, is dependent upon biomechanics, genetics, and metabolic health (Agarwal 2012; Botha, Lynnerup, and Steyn 2020; Cho and Stout 2003; Eleazer and Jankauskas 2016; Frost 1987). Features such as porosity and cortical density in bone histomorphometry have associations with age but are largely determined by genetic heritability and highly influenced by biomechanics, health, and the environment (Bjørnerem et al. 2015; Cho and Stout 2003; Havill et al. 2010).

As mentioned above, biomechanics play a large role in the remodeling habits of bone, particularly in weight bearing long bones but perhaps less so in the rib (Tommerup et al. 1993) and while the effect of metabolic stress on bone remodeling is still being investigated, malnutrition and disease are major contributors to the variation present in rib histomorphometry (Brenton and Paine 2007). It is for these reasons many bioarchaeologists are hesitant to estimate age using bone histology; however, it can be argued that macroscopic methods for age estimation suffer similar downfalls regarding macroscopic skeletal changes due to life history and pathology, but are still practiced regularly (Clark, Simon, and Hubbe 2020).

The effect of diet and disease on the outcome of histological age-at-death estimation in adults, was investigated by Brenton and Paine who estimated age-at-death from a population of known Black South African individuals from the Raymond A. Dart Skeletal Collection at the University of the Witwatersrand Medical School, Johannesburg, South Africa who were identified as having suffered from pellagra or general malnutrition (Brenton and Paine 2007; Stout and Paine 1992). Their results show that while individuals who suffer from general malnutrition are generally underaged (~24.4 years), while those who were specifically diagnosed with pellagra before death were underaged to a higher degree (~36.7 years). They recorded lower OPD, fewer secondary osteons, larger osteon and Haversian canal sizes, and less cortical area in the pellagra sample than those in the control population. Brenton and Paine note that the history of histological variances in nutritionally deficient populations are dependent on the type of metabolic or dietary disease and its effect on the accumulation of osteons in secondary compact bone (Eriksen et al. 1993; Robling and Stout 2003; Wu et al.

1970) and that nutritional status should be considered before practicing age estimation methods that were established using healthy individuals (Paine and Brenton 2006).

Although it seems bone histological changes occur in a predictable pattern in children and adults, the research shows that the "normal" rate of remodeling is affected by factors such as health and diet that impairs the functions of bone cells and causes a dysregulation in cellular coupling (Agarwal and Miller 2016; Paine and Brenton 2018; Streeter 2010). Since cellular responses to disease begin below the bone surface before they are observable macroscopically, histomorphometric examination of the rib may help bioarchaeologists identify the extent of poor health in individuals who do not exhibit pathological changes on the skeleton so that caution can be exercised when age at death estimation is performed (Paine and Brenton 2006; Pfeiffer 1998; Stout and Teitelbaum 1976). An expression of age associated variation within the histomorphometry of the Kilkenny Union Workhouse population sample is expected but may vary due to stress including disease, and malnutrition induced by the Great Famine.

2.5.3. DIRECT AND INDIRECT ANALYSES OF BONE HISTOLOGY

The cellular processes that regulate normal bone remodeling in response to growth, development, and mechanical stimuli with age associated patterns are sustained by paracrine or autocrine hormonal signaling, such as the parathyroid hormone which stimulates osteocytes to initiate osteoblast direct RANK ligand for osteoclast differentiation (McCarthy 2016). The regulatory power of osteocytes to signal for bone remodeling and the success of osteoblasts and osteoclasts is highly dependent on the bioavailability of key nutrients that are necessary for an adequate balance of calcium, phosphate, and collagen in bone (Brickley, Ives, and Mays 2020).

Osteocyte function can also be affected by elevated levels of pro-inflammatory cytokines due to inflammatory diseases including bowel disease, bacterial or viral infection, and rheumatoid arthritis (Zhou, Li, and Pathak 2019). This increase in inflammation can cause proliferation of bone loss and affect other soft tissue organs of the body (Zhou, Li, and Pathak 2019). Recent studies have shown that when the effect of mechanical stimuli is controlled for through the use of a mechanically stable element like the rib and growth-related changes are accounted for by grouping samples into age associated categories, bone histology techniques can be useful aids in investigating the effect of diet, nutrition, and disease in bone (Pfeiffer et al. 2006; Stout, Cole,

and Agnew 2019). If significant differences are observed in histomorphometric variables between individuals within the same age group, a disruption in the remodeling process related to the presence or absence of disease may account for the variation.

The goal of this thesis is to clarify how diet and disease induced by indirect violence is presented in bone histomorphometry. The previous two sections explained how bone histological variables are influenced by biomechanical processes and age. To inform the first, second, and third aims of this thesis, this section will describe how changes in diet, disease, and other stressors brought on by socio-political inequality may affect the processes of bone remodeling that can be viewed through the microscope.

2.5.3.1. Histopathology: The direct analysis of lesion histology

Analysis and identification of osseous changes potentially associated with metabolic or infectious diseases are typically observed and recorded from a macroscopic perspective (as described in Chapter 3). However, radiographic and histological analyses can provide support for differential diagnosis of disease (Assis and Keenleyside 2016; Brickley, Ives, and Mays 2020). Often, when bone histology is conducted to examine the effect of disease on human skeletal tissue or to aid in differential diagnosis, samples are taken directly from skeletal lesions (Stout and Teitelbaum 1976; Schultz 2001).

Early microstructural observations of bone lesions were described by Weber who observed histological changes in bone including bone resorption and cortical thickening for those afflicted with disease (Weber 1928). Later, Stout and Teitelbaum observed scalloped cement lines in the iliac crest of an individual with Paget's disease as well as numerous Howship's lacunae due to excessive bone resorption in an individual with evidence of osteomalacia (Stout and Teitelbaum 1976). The authors also identified woven bone in an older individual who may have suffered from renal disease, hyperparathyroidism, or another malady that causes increased resorption and quick deposition of bone (Stout and Teitelbaum 1976).

Schultz has asserted that light and polarized dry bone microscopy is an underused method of differential diagnosis and identified numerous features in lesion microstructure that can be used as supporting evidence in the identification of diseases present in the archaeological skeleton

(Schultz 2001; 2012; Schultz and Schmidt-Schultz 2015). In his work, remnants of chronic inflammation from periosteal new bone formation in microstructural form are referred to as Polster, new pillow-like bone formation, and Grenzstreifen, a band separating the original compact bone from the Polster (Schultz 2001).

In the same paper, Schultz also describes how the features of nutritional deficiency, such as iron-deficiency anemia, scurvy, rickets and osteomalacia, and other inflammatory diseases, are similar in their macroscopic pathological changes but can be distinguished by light microscopy (Schultz 2001). In one case, porotic hyperostosis in the skull presented as a non-specific symptom that may be indicative of either anemia and scurvy, but microscopic analysis differentiated the ectocranial lesion as a subperiosteal hematoma because it was situated on the original bone surface, the external lamina was not affected, and the structure of the diploë was normal. The presence of woven bone built up from fibrous connective tissue supported the hypothesis that the lesion occurred because of an ossified hematoma and the unaffected bone surface indicated the cause was Vitamin C deficiency (Schultz 2001). Schultz also suggested porotic hyperostosis of the vault can be diagnosed as rickets and osteomalacia if histological analysis showed thickening of the vault and the appearance of splintering of the internal and external lamina (Grauer and Buikstra 2019; Schultz 2001).

The proclamation of the utility of dry bone histological analysis for differential diagnosis of a host of lesions present in archaeological bone has led to new interest in bone histology. Von Hunnis and colleagues found evidence of Grenzstreifen and Polsters as well as sinuous lacunae indicating lytic activity in the bone microstructure of two skeletons with macroscopic evidence of syphilis, which supported their hypothesis regarding the presence of syphilis in pre-Columbian England (Von Hunnius et al. 2006). However, the utility of bone histology as a pathognomonic tool to accurately identify disease etiology has been questioned. For example, Weston tested the method using scanning electron microscopy on a sample of museum specimens from St. George's Hospital Pathology Museum in London, UK and found that samples with the histological features (Polsters, Grenzstreifen, and sinuous lacunae) were not diagnostic of any specific disease (Weston 2009). De Boer and colleagues emphasize the lack of specificity in Schultz' descriptions of histological features for rickets and osteomalacia and

call for more detailed research on fresh bone tissue diagnoses and experimental data (Schultz 2001; De Boer, Van der Merwe, and Maat 2013).

Unfortunately, the nature of bone tissue is such that it can only be removed or replaced and while there is conflict regarding the pathognomonic power of dry bone histology, the consensus is that few diseases show enough variation in cellular activity to differentiate between the array of specific and non-specific diseases that can affect the skeleton. To further complicate the possibility of diagnosing pathology using dry bone histology, histological symptoms of nutritional deficiency or disease can clear up shortly after the recovery process begins. For example, it is common for radiolucent scurvy lines in the metaphysis of long bones in children with Vitamin C deficiency to become obliterated with age associated remodeling or increased porosity (Brickley, Ives, and Mays 2020). For now, attempts at differential diagnosis should always include macroscopic evidence when it is available (Brickley, Ives, and Mays 2020; De Boer, Van der Merwe, and Maat 2013; Boer and Merwe 2016; von Hunnius et al. 2006; Weston 2009).

2.5.3.2. Systemic changes in bone histology: Indirect analysis of the histological effects of disease

Although histological analysis of samples cut directly from lesion sites can support specific diagnoses and help researchers understand the healing process, these changes are the result of underlying bone cell activity which may cause systemic changes in bone remodeling throughout the skeleton (Cho and Stout 2003; Ortner and Putschar 1981). Indirect histomorphometric analysis of these changes can provide a general idea of the biological and cultural factors that affect bone remodeling and influence the outcome of bioarchaeological interpretations of past populations without destroying lesions (Boivin and Meunier 1993; Chan et al. 2020).

Bone histology has been used to indirectly infer bone health through the interpretation of histomorphometric features in the femur, rib, metacarpals, vertebrae, and other elements (Brickley and Agarwal 2003; Chan et al. 2020; Cho and Stout 2003; Martin and Armelagos 1985; Velasco-Vázquez et al. 1999). In clinical and archaeological studies, bone health is often determined by the extent of normal, age-related osteoporosis or secondary osteoporosis, which can occur earlier in life as a result of any range of disorders (Brickley and Agarwal 2003; Stein

and Shane 2003). Osteoporosis is a general non-specific trait indicative of older age, hormonal imbalance, disuse, or poor health that is defined by its low bone tissue volume and identified by the presence of multiple bone resorption spaces (Agnew and Stout 2012; De Boer, Van der Merwe, and Maat 2013) which can lead to skeletal fragility and increase the risk of fracture (Christodoulou and Cooper 2003; Mays, Turner-Walker, and Syversen 2006).

Age is a common cause of osteoporosis in older adults, but secondary osteoporosis, or thinning of the cortical bone due to disease, can occur when there is an imbalance in the remodeling process (Beresheim, Pfeiffer, and Alblas 2018; Brickley and Agarwal 2003; Martin 1981). While osteoporosis is not pathognomonic of any specific disease, it is a symptom of many ailments and studies into osteoporotic bone are helpful for understanding the sequence of bone remodeling in individuals affected by general malnutrition, disease, and other factors related to social inequality (Chan et al. 2020; Cho and Stout 2003; Garn et al. 1971; Pfeiffer and King 1983). Recently, Chan and colleagues found that risk of osteoporosis was associated with lower monthly income and lack of formal education for adult women in China (Chan et al. 2020). Additionally, a past study showed low percent cortical area was due to low calcium intake, calcium to phosphorus ratio, and protein-calorie malnutrition partially attributed to a primarily corn-based diet in two protohistoric Iroquoian population samples, the Kleinburg and Uxbridge groups, from AD1600 and AD1490 + 80, respectively (Pfeiffer and King 1983).

Porosity and cortical bone density were also indicative of poor health in adults in a study on the impact of post-reconstruction lifestyles by Martin, Magennis, and Rose (1987). These researchers explored the bone microstructure of “Afro-Americans” from an unmarked Baptist Church cemetery in Cedar Grove, Arkansas and discovered low cortical area and high porosity correlated with high frequencies of skeletal lesions that were indicative of extremely low nutrition and rampant infectious disease (Martin, Magennis, and Rose 1987). In this study, the authors used a biocultural investigatory approach (see Section 2.5.4) that included research into local historical literature, oral histories, and biological anthropology to piece together the life experience of the African Americans who were buried in the unmarked graves. This study showed that bone histological analysis can be effective for understanding the biological consequences of social and political hierarchy and demonstrates the importance of placing skeletal research of any modern or archaeological population into its appropriate social context.

When greater emphasis is placed on gathering histomorphometric data in samples from populations with documented stress events, bioarchaeologists can gain a better understanding of the circumstances of health in ancient or historical populations without contextualized pasts. For example, Robbins Schug and Goldman (2014) found that young children with shorter stature from the Late Jowre phase of Prehistoric India (1000-700 BC) did not maintain or acquire the bone mass expected for their age. While this group had fewer paleopathological lesions, bone histological analysis indicated the presence of gross lesions as evidence of frailty should be critically evaluated.

In another study, Brenton and Paine compared macroscopic features between adults from the Raymond Dart Skeletal collection known to have pellagra (n=14) and individuals with general malnutrition (n=17) and found those with pellagra had higher bone alveolar loss, dental caries, enamel hypoplasias, periostitis lesions, osteomyelitis, cribra orbitalia, and cranial pitting than those with general malnutrition (Brenton and Paine 2007). These macroscopic features differentiated the pellagra group from the general malnutrition group but were not specific to pellagra. They also used this sample to compare bone histology between the disease types (pellagra n = 10; general malnutrition n= 14) and found those with pellagra exhibited decreased cortical area compared to their age associated peers with general malnutrition. The results from this study indicate that bone maintenance in individuals with extensive niacin deficiency may be affected to a greater degree than bone maintenance in individuals with general malnutrition (Brenton and Paine 2007).

Psychosocial stress affects can cause an imbalance in bone remodeling due to the impact on glucocorticoid levels (Suarez-Bregua et al. 2018). Glucocorticoid levels play a large role in osteoblastogenesis and recent research shows that increased levels can cause imbalances in bone homeostasis (Schiavone et al. 2016) including reduced bone accumulation, increased bone resorption, decreased bone mineral content, or hyperostosis in animal models that have not been treated with anti-psychotic medications (see: Suarez-Bregua et al. 2018). Since many of the archaeological populations discussed in this thesis, including the study population, are marginalized populations that lived in poverty and suffered from disease and malnutrition, it is likely they experienced the skeletal effects of psychosocial stress.

While cortical area and porosity are of primary importance for evaluating the quality of bone maintenance, bone health can also be interpreted by observing the histological features that change in a predictable pattern during the natural process of bone remodeling in cortical bone, including the quantification of osteon and Haversian canal size and shape and accumulation of osteons within the cortex (Burr, Ruff, and Thompson 1990; Goliath, Stewart, and Stout 2016; Stout, Cole, and Agnew 2019). Uncoupling of the bone remodeling process may reduce the rate of osteon formation, resulting in a larger mean osteon area and less circular osteons in more metabolically stressed individuals. Since Haversian canals are associated with osteon size and shape, these features will also be affected. For example, Pfeiffer found less variation and smaller Haversian canals in the ribs compared to their femora of individuals from the nineteenth-century Spitalfields collection. Pfeiffer states that they find this is not consistent with the assumption that Haversian canal area reflects whole body metabolic activity (Pfeiffer 1998). In this paper Pfeiffer did not address the capability of ribs to reflect more recent changes in metabolic processes (Epker and Frost 1965), but she does mention the potential for Haversian canals to be informative about metabolic health and suggests future studies should focus on the rib due to its small cross section (Pfeiffer 1998).

2.5.4. BONE HISTOLOGY AND THE BIOCULTURAL APPROACH

In the archaeological record, the skeleton is the biological connection between material culture, such as tools, burial goods, and burial practices, and the physical body (Quinn and Beck 2016). To better understand the relationship of the physical body in death to its living identity, bioarchaeologists examine skeletal markers related to health and disease, but to deeply explore the life experience of people from the past, it is important to center the skeleton within the context of its historical, cultural, and environmental background using cross-disciplinary methodology, theory, and data (Goodman et al. 1988; Zuckerman and Martin 2016). In bioarchaeology, this is known as a biocultural approach.

Bioarchaeologists benefit from a biocultural approach to skeletal analysis in that they can better interpret biological variation in the skeleton by understanding how humans have adapted to their changing environments and how they may have experienced stress due to political, climate, or economic crises, all of which have affected human variation over time (Agarwal and

Glencross 2011; Buikstra and Beck 2006; Leatherman and Goodman 1997). However, this holistic method of approaching research from a multidisciplinary perspective has not always been the default in the field of anthropology and the consequences of past racial typology (i.e., Morton 1839; Beddoe 1870; Howells 1941; Hooton and Dupertuis 1955) and descriptive approaches to skeletal analysis still impact the field and society today (Armelagos 2008; Boas 1904; Buikstra and Beck 2006; Goodman et al. 1988; Larsen 2018; O'Donnabhain and Murphy 2014). To understand the significance of biocultural themes and methodologies in the field of anthropology, Agarwal and Glencross have contextualized the progression of engagement with the approach as having occurred in three waves(Agarwal and Glencross 2011).

2.5.4.1. The First Wave of Biocultural Bioarchaeology

The first wave of incorporating biocultural methods into bioarchaeological research is characterized by the acknowledgment of the ability of the skeleton to adapt to environmental forces. This variation on Wolff's Law, which describes how the skeleton adapts to differences in loading environment (Wolff 1892; Frost 1990), prompted investigations into human adaptation to stress, disease, biomechanics, growth and development, and trauma (Agarwal and Glencross 2011; Goodman et al. 1988; Goodman and Leatherman 2020; Knüsel 2010; Temple and Goodman 2014). When biological anthropologists began considering biocultural concepts into their research, the aims drifted from typological and descriptive case reporting reminiscent of the categorical focus of Linnaeus and Darwin to more hypothesis driven research (Armelagos et al. 1982; Armelagos et al. 2010; Zuckerman and Armelagos, 2011; Zuckerman and Martin 2016).

Early examples of this initial wave include Lawrence J. Angel's paper in the *American Journal of Physical Anthropology* from 1946 that used biodistance analysis to explore the degree of heredity between the Mycenaean and Classic Greek cultures with the aim to better understand their social interactions (Angel 1946; Buikstra, King, and Nystrom 2003). Arriving on the heels of this manuscript and supported others who had recently begun to criticize the typological approach to osteological analyses (Boyd 1950; Buikstra, King, and Nystrom 2003; Stewart 1954, Buettner-Janusch 1969), was the incorporation of the concept of a "New Archaeology" paradigm (Binford 1968; Darvill 2008) into the psyche of bioarchaeologists in what Washburn referred to as the "New Physical Anthropology" (Washburn 1951). In this processual approach

to anthropology, ecological, cultural, and social factors are included in the study of archaeological sites and populations (Tuttle 2018).

One of the most groundbreaking examples of the use of the biocultural approach in bioarchaeology was conducted by Livingstone who explored the relationship between the mosquito infested agricultural environment of West Africa and the prevalence of selection of the heterozygous sickle-cell gene that protects against malaria (Livingstone 1958). By incorporating genetic, environmental, entomological, and cultural considerations into the investigation of the sickle-cell gene, Livingstone demonstrated the promise of intersectional research to reconstruct patterns of human adaptation and, in this case, dispel notions of "racial markers" (Armelagos 2008; Baker and Agarwal 2017; Livingstone 1958).

Livingstone's study was also one of the first to describe the environment as more than just one's physical surroundings through what is referred to as "ecological anthropology", a theory of the environment as any ecological, social, or biological entity that can affect the human behavior and biology (Leatherman and Goodman 1997; Zuckerman and Armelagos 2011; Zuckerman 2016). The impact of the environment, whether positive or negative, can be affected by cultural buffering mechanisms that relieve the impact of stress in terms of any physiological disruption resulting from insult (Goodman and Leatherman 2020), or act as agents that can magnify or cause stress. Understanding the dialectic nature of biology, environment, and culture adds nuance and complexity to the stories we tell about humans and societies from the past and incorporating a biocultural approach into research can help untangle these tales in an engaging way.

Bone histological analysis is not often discussed as a methodology that contributed to the development of biocultural approaches in bioarchaeology. Like many osteological studies, early papers by Harold Frost (1921-2004), a pioneer in bone histological research, and other bone biologists were mainly descriptive reports on bone loss and remodeling parameters (Amprino 1948; Epker and Frost 1965; Frost 1969; Jaffe 1929; Wu et al. 1970). Later, Frost began investigating ways to control for age and biomechanically associated factors that are inherent in bone remodeling patterns to understand the effect of disease, nutrition, occupation, and other social and environmental influences on changes in bone histological features (Frost

1987). Other bone histologists also began looking at changes in cortical bone histology that can be associated with these influences (Burr, Ruff, and Thompson 1990; Martin and Armelagos 1985; Martin, Magennis, and Rose 1987; Richman, Ortner, and Schulter-Ellis 1979).

For example, Burr and colleagues used bone histomorphometry to look at culturally based secular change related to lifestyle between an archaic Pecos Indian sample and a modern sample and found that smaller Haversian canal size and greater OPD in the Pecos sample was likely to indicate a more active lifestyle for the archaic group (Burr, Ruff, and Thompson 1990). A previous study examined the differences in histomorphometry between three early American populations, the Eskimo, Arikara, and Pueblo, and found differences in the presence of Type II osteons, which they correlated to protein intake and physiological well-being (Richman, Ortner and Schulter-Ellis 1970). Many investigations into the clues bone histology can provide about the impact of diet and role of society in the quality of bone health have been conducted since this transition into bioculturally minded research and the subject continues to be a topic of interest today (Maddalozzo et al. 2009; Marklein and Crews 2017; Mays, Turner-Walker, and Syversen 2006; Miszkiewicz and Cooke 2019).

The first wave of biocultural research in bioarchaeology was important because as anthropologists and scientists, the conclusions of our research carry a lot of weight in the eye of the public. The contribution of biological anthropology to the eugenics movement, for example, is a part of the field's history that had enormous consequences regarding the way race is perceived throughout the world and continues to affect the lived experiences of marginalized people today (Levine 2010). The hypotheses laid out in Chapter 1 follow in the footsteps of this first wave of biocultural research and aim to contextualize histomorphometric methods with the experience of poverty in nineteenth century Ireland. The intent is to contribute to the understanding of the reaction of bone cells to disease and malnutrition through the analysis of a group of people who died because of the wretched social conditions in which they lived.

2.5.4.2. The Second Wave of Biocultural Bioarchaeology

The second wave in biocultural bioarchaeology is described by Agarwal and Glencross as two different yet converging groundbreaking concepts (Agarwal and Glencross 2011). The first is the utilization of innovative technology in skeletal biology methods. This includes the use of

isotopes (Miller et al. 2020a; Reitsema and Holder 2018; Vogel and van der Merwe 1977), genetic analyses (Eerkens et al. 2016; Hill 2000; Knudson and Stojanowski 2008), and non-destructive micro-imaging techniques (Maggiano et al. 2016). While each of these methods have allowed bioarchaeologists to ask large, population-level questions using macro and nano-scale data, Agarwal and Glencross have argued that even these data can sometimes fall into the descriptive pattern without proper analytical focus (Agarwal and Glencross 2011).

Many bone histological studies have been conducted in conjunction with the technologies mentioned above. For example, isotope analyses have helped to reveal more about the relationship between diet and bone remodeling (Arnay-de-la-Rosa et al. 2011; Fahy et al. 2017) and powerful imaging tools such as synchrotron analysis have revealed new avenues for three-dimensional investigations into the histological structure of bone (Maggiano et al. 2016). The second wave is also characterized by the recognition that skeletal samples are not unbiased representations of the populations from which they are derived. This concept became widely acknowledged with the publication of Wood and colleague's manuscript "The Osteological Paradox". The theory of the osteological paradox considers the nature of the archaeological skeletal record as complex assemblages that exist due to selective mortality and varying degrees of individual frailty (Wood et al. 1992). This innovative way of thinking about skeletal analysis caused an even greater shift in the transition from descriptive analysis toward hypothesis-driven research (Boldsen and Milner 2012; Grauer 2018).

Since the results of the isotopic analysis of individuals from the Kilkenny Union Workhouse burials will be incorporated into this thesis, a summary of the basis of isotopic research, a key technological advancement in the second wave of biocultural approaches to a social bioarchaeology, will be presented. Then, a review of the osteological paradox will be incorporated to demonstrate the significance of the theory in the field of bioarchaeology and provide background for the research design of this thesis.

Stable Isotope Analysis: When humans consume plants and animals, the carbon and amino acid proteins from those food sources are transferred into the organic collagen and inorganic hydroxyapatite matrix of the skeleton so that the carbon and nitrogen content reflect the isotopic composition of their diet (Ambrose and Norr 1993; DeNiro and Epstein 1981; Tieszen and

Fagre 1993). These stable isotope values can be retrieved from dental enamel, dentine, cementum, and calculus, as well as bone collagen, hair, nails, skin, and muscle using a mass spectrometer to read the record of life history in humans—known as "the tissue clock" (Fahy et al. 2017; King et al. 2018; Neuberger et al. 2013; Scott and Poulson 2012; White, Longstaffe, and Law 2004). Miller and colleagues describe the advantages of this biological clock by its ability to examine "relationships between food access and variables such as sex, gender, age, social status, and biological processes such as how diet and nutrition influence growth, development, and disease" (Miller et al. 2020a: 568).

Analysis of carbon ($\delta^{13}C$) and nitrogen ($\delta^{15}N$) isotope ratios provides a plethora of information regarding the experience of early life, mid-life, and near death for individuals and communities as a whole. Values for $\delta^{13}C$ and $\delta^{15}N$ derived from body tissues are based on photosynthetic food type differences and trophic level of the consumer (Ambrose 1991; Winagawa and Wada 1984; White, Longstaffe, and Law 2004). Terrestrial plants, for example, are identified based on how much atmospheric $\delta^{13}C$ they exude during photosynthesis. Grains, trees, fruits, and other vegetables are C_3 plants and incorporate the least $\delta^{13}C$ while C_4 plants such as maize, millet, and other grasses incorporate more $\delta^{13}C$. Protein heavy diets will be represented as $\delta^{15}N$ isotope values, usually indicating the presence of legumes, aquatic animals, or other meat in the diet. While it is widely accepted that "you are what you eat", some studies have suggested that the variation between foods consumed and $\delta^{13}C$ and $\delta^{15}N$ values varies more than is understood and further validation studies are warranted with local isotopic baselines for the environment under investigation (Gannes, O'Brien, and Rio 1997; Goude and Fontugne 2016).

In bioarchaeology, isotopes have been useful for revealing periods of stress in adults and children from the past. Recent research shows that the life experience of individuals suffering from dietary stress will be reflected in the $\delta^{13}C$ and $\delta^{15}N$ values of their bone and dentine. These biocultural studies of humans from the past can inform how poverty and starvation was experienced in different cultures across time and space (Eerkens and Bartelink 2013; Miller et al. 2020a; Reitsema and Vercellotti 2012; White 2005). For example, a study that looked at the impact of eating disorders on $\delta^{13}C$ and $\delta^{15}N$ isotopes from hair found high $\delta^{15}N$ values in those with poor diets (Hatch et al. 2006). Geographic relocations, drought, flooding, and other environmental stressors that prevent the regular cultivation of crops have also been found to

influence $\delta^{15}N$ values (Miller et al. 2020a; Redfern et al. 2019; Vogel and van der Merwe 1977); while the reliance on corn as a primary form of subsistence has been shown the alter bone porosity (Paine and Brenton 2007). Alternatively, a study comparing the trabecular bone porosity between groups from pre- and post-Spanish conquest showed no difference in osteoporosis that could be attributed to colonization, suggesting that the observed changes in stable isotopes were not due to malnutrition (Arnay-de-la-Rosa et al. 2011). Biocultural approaches that combine isotope data with historical records of people from the past can help guide inferences about the variation of life experiences of on sex, age, gender, social status, and other socio-political factors that may have influenced health.

One attempt by the British Government to relieve the poor of starvation was to import Indian meal (maize) into the country from North America (see Section 3.3.1.). Since the workhouses in Ireland were notorious for their differential treatment of inmates based on class, sex, and age in terms of who was accepted for indoor relief as well as labor and resource distribution within the workhouse, it is possible that relief food was allocated with varying preference for some groups over others (Geber and O'Donnabhain 2020). To determine if resource distribution varied amongst the victims of the Famine interred in the Kilkenny Union Workhouse mass burial ground, Beaumont and Montgomery analyzed the stable isotope ratios of rib bone and dentine for evidence of relief food in the form of maize, a C_4 plant, as indicated by values lower than -17‰, and evidence of starvation, as indicated by $\delta^{15}N$ isotope values higher than >12‰ (Beaumont et al. 2013; Beaumont and Montgomery 2016). The authors reported a change in $\delta^{13}C$ between the dentine and the rib bone collagen, indicating a transition from C_3 potatoes to C_4 maize from childhood until just a few years before death. High levels of $\delta^{15}N$ also indicated stress throughout life. Both dental enamel and rib bone collagen confirmed the long-term nutritional and physiological stress endured by the people who experienced the Famine before and during their time in the workhouse (Beaumont and Montgomery 2016). These results showed that the change in diet could be observed through isotope ratios and that stress has an impact on the interpretation of $\delta^{13}C$ and $\delta^{15}N$ profiles, which may make interpretation difficult in populations that are not so heavily contextualized.

One way to tie together the information about diet with paleopathological data and historical context is to consider how isotope analysis can inform us about the presence of diseases that may not be mentioned in the historical record but would have existed in the population under study, nonetheless. While illnesses such as typhoid and tuberculosis were frequently identified during the Famine, the diagnoses could only cover so much as the history of medicine would allow. Therefore, diseases we know of today are not present in the historical medical and census records. One such disease is pellagra, a condition that more than likely would have been present in the workhouse but would not have been recognized as such during the Famine due to the lack of awareness about the vitamin deficiency, much less its etiology. The identification of pellagra was made even more difficult by its similarity to other diseases which were documented during the Famine. The following section describes pellagra and its clinical manifestations.

The Manifestations of Pellagra:

In the palaeopathological analysis of skeletons from impoverished, migrant, and/or starving population samples, pellagra—if mentioned—is commonly discussed as a condition from which many people likely suffered. However, differential diagnosis of pellagra, like many diseases, is difficult as the symptoms associated with the condition can cause lesions similar to those of other disorders (Brickley and Ives 2008). Pellagra is a nutritional deficiency disease associated with a lack of Vitamin B3, or niacin, which is essential for the production of the nicotinamide coenzyme (Miller et al. 2020b). When the body is deficient in niacin, nicotinamide catabolism is disrupted, which can cause impairment of cellular energy metabolism and DNA repair (Mariani-Costanini and Mariani-Costanini 2007; Miller et al. 2020b). While niacin can be produced endogenously through the synthesis of the amino acid tryptophan, the conversion cannot take place if Vitamins B2 and B6 are also deficient from the diet (Crabb 1992; Segula et al. 2012). The daily recommended intake of niacin is 15-20mg. If ingestion of niacin and tryptophan are lower, or if the body cannot synthesize or metabolize the amino acids, then pellagra will develop (Etheridge 1972; Segula et al. 2012).

In living people, a diagnosis of pellagra is made by taking a full social and dietary history of the patient as well as conducting a physical examination (Segula et al. 2012). A patient may be suffering from primary pellagra if the diet is not diverse enough to include niacin or tryptophan

rich foods such as yeast, eggs, bran, peanuts, meat, poultry, fish, legumes, or whole grain cereals, or if other diseases like alcoholism prevent the body from properly absorbing niacin (Badaway 2014; Itani et al. 2019).

The earliest description of pellagra was recorded by Don Gasper Casal in 1735 as “mal de la rosa”, or sickness of the rose, in English (Roberts 1990). He referred to the symptoms of pellagra as the “Four Ds,” dermatitis, diarrhea, dementia, and, if untreated, death. In patients who were frequently suffering from chronic alcoholism, gastrointestinal diseases, or malnutrition, Casal observed a red and glossy rash on sun exposed areas of their bodies, primarily the hands, feet, and neck (known as Casal’s Necklace). The rashes, known as dermatitis, were frequently accompanied with thickened skin, inflammation of the mouth and tongue, and episodes of diarrhea. Mental fatigue, irritability, anxiety, depression, and forgetfulness were also described along with other symptoms of neuropathy resulting in both physical and mental states of distress for the patient (Brenton 2009).

Where access to supplemental vitamins and proper nutrition are available, those suffering from pellagra can easily recover (Kertesz 2001). However, in populations with little to no access to diverse food sources, recovery is more difficult. Ironically, when pre-treated with alkali ingredients through a cooking process called nixtamalization, niacin becomes bioavailable and can be somewhat sustainable; but during the Great Famine, those in the workhouses subsisted primarily off of unprocessed maize, leaving them highly vulnerable to pellagra (Segula et al. 2012; Beaumont and Montgomery 2016).

One of the only known documented skeletal cases of death by pellagra are fourteen individuals from the Raymond Dart Skeletal Collection housed at the University of Witwatersrand Medical School in Johannesburg, South Africa. Paine and Brenton (2006) examined these skeletons for differences in the macro and microstructure to individuals with pellagra and those who died from non-specific malnutrition as well as two individuals with scurvy. They examined osseous lesions for each individual as well as alveolar bone loss and cortical bone loss for the rib. In this study, those with pellagra exhibited high frequencies of lesions similar to those seen in scurvy and iron deficiency anaemia, particularly in the lower leg. Those with pellagra also

experienced greater alveolar bone loss and cortical bone loss of the rib though these results were not significantly different between causes of death. The authors determined pellagra and non-specific general nutrition differentially affects bone histological features and osteon population density (described in Chapter 4), and may help to elucidate dietary problems of historic and prehistoric communities (Paine and Brenton 2006).

In another study, Miller and colleagues (2020b) examined a small group of people known to have died with pellagra in the Ferrara psychiatric hospital in Italy (1830-1870). Paleopathological analysis of patients known to have pellagra showed high prevalence of periodontal and other dental diseases as well as many non-specific disease indicators on the skeleton including pitting and deepening of the meningeal grooves, cranial asymmetry, and cribra orbitalia. Ectocranial porosity was observed on the left orbital, glabellar, and maxilla regions, and endocranial porosity was also observed on the sphenoid, temporals, and occipital. Enlarged mandibular mental and mylohyoid foramina were present and atrophy and hyperostosis of the pituitary fossa was recorded. These individuals showed low rates of lesions indicative of infectious disease, despite records that state many patients suffered from infection (Miller et al. 2020b). This study emphasized the biocultural implications of pellagra as a disease of the poor and the toll that stigma had on the progress of diagnosis and preventative measures that would allow the epidemic to cease once it was better understood.

In this thesis $\delta^{13}C$ isotope values in the C_4 range, indicative of maize consumption, will represent individuals who likely suffered from pellagra. This information will help guide the discussion regarding whether the presence of pellagra, a metabolic disease, affected bone remodeling.

The Osteological Paradox: As previously stated, pathological analysis of the skeleton is essential for understanding the health of past populations. The simplicity in that statement, while it holds much truth, harkens back to what is now referred to as the "conventional wisdom" approach to interpreting pathological changes in skeletons recovered from archaeological assemblages (DeWitte and Stojanowski 2015). In the conventional wisdom approach, osteological stress markers (OSMs) such as bony lesions, enamel defects, Harris lines, and

others are interpreted as indicators of overall health or lack thereof (i.e., the higher the frequency of these OSMs the more stressed the population must have been) (Sołtysiak 2015).

Use of the conventional wisdom approach to archaeological skeletal samples implies that skeletons are reasonable representations of the living populations they were once a part of and that the pathological changes observed are true representations of the health of those populations (Cohen, Wood, and Milner 1994). While this seems logical enough, the act of interpreting population health from a sample of deceased individuals is inherently contradictory. In 1992, Wood, Milner, Harpending, and Weiss published a seminal paper, “The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples" addressing this very concern (Wood et al. 1992). In their paper, Wood and colleagues state that the problems inherent in using samples of deceased individuals to infer population health have to do with three main issues: demographic non-stationarity, selective mortality, and hidden heterogeneity (Wood et al. 1992).

Demographic non-stationarity addresses the concern that cemetery or burial assemblages, especially those that were in use for long periods of time, may be composed of individuals from populations that experienced migration or temporal changes. Stationarity in a population with little influence from migration ensures consistency in rates of fertility, mortality, and age distribution throughout the sample. Stationarity also controls for the rate of population growth, which affects all other variables. In populations that experience non-stationarity, Wood and colleagues emphasize that age distributions in burial samples reveal more about levels of fertility within the group rather than mortality patterns, which can confound the results to bioarchaeological questions regarding group susceptibility to death (Wood et al. 1992).

In historic populations, using biocultural research methods such as the inclusion of archival data (i.e., census and burial records, newspapers, first-hand accounts) that recorded the rate of migration to or from the region under study and indicate how long the burial site was in use, can help mitigate the issue of demographic non-stationarity. However, sites from the more distant past may not have access to historical records such as these. Fortunately, detailed population statistics about those who entered Kilkenny City and the Kilkenny Union Workhouse prior to and during the famine, including age, sex, migration, and number of births

and deaths, were reported in the 1841 and 1851 census records (Census of Ireland Commission 1843; 1853) and the minute books kept by the Kilkenny Board of Guardians, which are now housed at the Kilkenny County Library.

Selective mortality is an issue inherent in all skeletal samples from which population health is being assessed. When bioarchaeologists use skeletal samples to learn about the life of past people, they are doing it from the perspective of the dead. Therefore, our mortality reflects the burial record and, more specifically, those whose skeletons are excavated. According to Wood and colleagues, the sample under study while once alive, is representative of the people who died and therefore, cannot be completely representative of the health or increased risk of death of the population. In other words, those who died with skeletal lesions are only representative of those who did not survive and not the actual count of those who were at risk but survived and died of another cause later in life. This makes establishing hypotheses regarding the relative risk of death for specific demographic groups more complicated since skeletal samples will always be biased representations of the community they once belonged to (Boldsen 2007). However, in the Kilkenny Union Workhouse sample it can be assumed that all individuals were at risk of death, since only those who were in this state of well-being were accepted into the workhouse. Therefore, those who were buried on the grounds of the workhouse are more closely representative of the workhouse population and, as a proxy, the poor in Ireland during the Great Famine.

Hidden heterogeneity or heterogeneity in frailty is the concept that individuals are unequal with respect to their susceptibility to disease and other stressors, and therefore, their risk of death (Wood et al. 1992). The idea is that these genetic and cultural variations in the susceptibility to disease are inherent in living populations and difficult to tease out in skeletal samples, they are "hidden" (Wood et al. 1992). Differences in genetics, socioeconomic class, sex, age, and many other factors like underlying health issues such as immunodeficiency diseases can cause variations in frailty that are hidden in the osteological record (Cohen, Wood, and Milner 1994; DeWitte and Bekvalac 2011). However, all individuals that exist within a burial sample were frail to some extent in life unless they died as a result of trauma, and even those who have resisted stressors over long periods of time might exhibit evidence of frailty (Marklein, Leahy, and Crews 2016). In fact, these individuals may express higher prevalence of OSMs due to their

prolonged exposure to stressors which creates a mortality/morbidity paradox in the skeletal record (Jackes 1993; Wood et al. 1992).

Perhaps the most controversial and complicated aspect of hidden heterogeneity in frailty is the question of the meaning of skeletal lesions. While documenting skeletal changes indicative of disease can help identify what types of stress a population suffered from, physiological responses to nutritional stressors are not always visible in the skeleton and observations of these lesions may not provide an accurate account of the experience of disease at the time of death. For example, lesions require time to develop and it is possible that someone with a weakened immune system may not have as much survival time as someone with stronger innate or adaptive immunity and more time to develop evidence of the disease on their skeleton (Cohen, Wood, and Milner 1994; Wood et al. 1992).

There is no doubt that those who were buried on the grounds of the Kilkenny Union Workhouse experienced short-term exposure to stress. However, the poor in Ireland survived many food shortages in the years before the prolonged Great Famine but many people failed to weather this event as they had in the previous seasons of crop failure. The representation of heterogeneity in frailty for those in the workhouse is potentially indistinguishable given the genetic, social, and cultural homogeneity of the population, but differences in susceptibility to disease may be observed based on age, sex, or hierarchical status prior to the continued failure of the potato crop throughout the Great Famine. Observations in remodeling events using bone histological analysis of the rib may inform whether variations in frailty prior to the famine can be better understood through the analysis of bone microstructure and disease presence.

Although their paper gained notoriety for challenging the conventional wisdom approach to skeletal lesion analysis and revealing key issues in interpreting population health from skeletal assemblages, Wood and colleagues were not the first to question the traditional methods of thinking. Demographic non-stationarity, survivorship, and lesion manifestation timing had been previously addressed by others (Angel 1975; Harpending and Pennington 1991; Ortner 1991; Paine 1989; Stuart-Macadam 1992) and in 1982, Bocquet-Appel and Masset published “Farewell to Paleodemography”, an essay about the inadequacies of age estimation in

establishing demographic profiles of past populations based on skeletal assemblages alone (Bocquet-Appel and Masset 1982).

Not all researchers were satisfied with this new theoretical concept, however, and many authored rebuttals to the osteological paradox (Cohen, Wood, and Milner 1994; Goodman 1993; Saunders and Hoppa 1993). For example, a reevaluation of the effects of sedentism and the adoption of agriculture on the population health of the Dickson Mounds people in Illinois from the perspective of the osteological paradox suggested that the increase in OSMs in farmers compared to earlier hunter-gathers may mean farmers experienced greater resistance to pathogens and had longer lives (Cohen 1989; Wood et al. 1992). This new assessment of their findings was rejected by Cohen and colleagues who contended that biological stress increases with farming, a hypothesis that was supported by extensive research including clinical data of individuals living with linear enamel hypoplasias (Cohen, Wood, and Milner 1994). Additionally, Jackes believed that Wood and colleagues were too generous in claiming there has been resolution to some questions in bioarchaeology including the issue of age at death estimation, but the author agreed that it was time to discuss the osteological issue many had been considering for a long time (Jackes 1993).

Wood and colleagues included suggestions for interpreting skeletal assemblages that acknowledge and mitigate the effects of the osteological paradox (Wood et al. 1992; Wright and Yoder 2003). These suggestions have been emphasized and elaborated on by more recent publications (Wright and Yoder 2003; DeWitte and Stojanowski 2015). The researchers suggest investigating the sources of heterogeneity in frailty. This can be done by leveraging archaeological contexts to better inform health inferences and focusing on “simple societies” with non-complex, egalitarian structures, and “simple sites” or those with short use histories and well-recorded histories (Wright and Yoder 2003; DeWitte and Stojanowski 2015). Referencing site background through life-history studies such as these help to reduce the effects of heterogeneous frailty if archaeological sites are determined to be culturally and biologically homogenous, consisting of one social group, and relatively egalitarian. If cemetery or burial sites were used for short periods of time by homogenous communities, then concerns with non-stationarity will be limited and relationships between health and demographics like age and sex can be more assessed more confidently.

Another suggestion for mitigating the effects of the osteological paradox is to focus efforts of analysis into age structured subgroups—specifically, into the effects of health and disease in subadults. Some studies have found that focusing on subadults is ideal for understanding frailty in the study population (Storey 1997). For example, young children are considered the most vulnerable individuals in a population and can serve as a control for frailty in samples with high frequencies of lesions for this age group (DeWitte and Stojanowski 2015). DeWitte and Stonjanowski explain that if there are more lesions in the vulnerable group, then the conventional wisdom approach can be supported, but if the older children have more lesions then this exemplifies the paradox (DeWitte and Stojanowski 2015). For example, one study found survivorship to be greater in groups with high lesion frequency in older children (Wright and Chew 1998), but another found higher prevalence of lesions in a lower socio-economic group than in the higher group of the same age (Bennike et al. 2005). Fortunately, new advances in technology-based methodologies such as isotope analysis can provide information about subadult diet and weaning practices that may give indications of early childhood health and conditions of frailty in subadult groups (Halcrow and Tayles 2008; Littleton 2011).

The third task biocultural anthropologists should consider when investigating individual and population health is leveraging the associations that can be made between skeletal lesions and other OSMs with demographic information while considering the effects of transgenerational health outcomes. This can be done through hazard modeling, a statistical model that allows the researcher to examine relationships between skeletal stress markers and mortality risk, life history interpretations, and indexes of skeletal frailty (Boldsen, Milner, and Weise 2015; Byers 1994; DeWitte and Wood 2008; Marklein, Leahy, and Crews 2016; McFadden and Oxenham 2020; Redfern and DeWitte 2011; Wilson 2014).

The final recommendation is the examination of the etiology and physiology of skeletal lesion formation. Bioarchaeologists have tackled this issue in their research into the processes of skeletal pathological changes and their meanings, whether the generated lesions are metabolic or infectious in nature, and how indicative lesions are of individual frailty in terms of resilience and recovery (Kyle et al. 2018; McFadden and Oxenham 2020).

Whether the osteological paradox theory is favored by the researcher or not, DeWitte and Stojanowski (2015) stress that failing to address the osteological paradox in modern bioarchaeological studies, particularly when it pertains to population health is a return to descriptive anthropology and a lazy approach to trying to understand human history. In fact, since the groundbreaking work by Wood and colleagues, recognizing the issues raised by the osteological paradox in research that involves interpreting skeletal lesions has become at minimum a consideration to keep in mind but sometimes a major aspect of investigation (Wood et al. 1992). Many researchers have added their own thoughts to the discussion (Byers 1994; Jackes 1993; Kyle et al. 2018; Reitsema and McIlvaine 2014; Sołtysiak 2015; Wright and Yoder 2003).

Byers contributed to the discussion by developing a methodology for determining whether stature is strongly selected for, which would refute the claim by Wood and colleagues that small stature may indicate less exposure to stress rather than more stress (Byers 1994). More recently, Kyle and colleagues explored the osteological paradox by comparing a sample of civilians to a sample of soldiers from Greek Himera. The authors predicted the soldiers, who were presumably leading healthier lives than the civilians, would have a lower prevalence of skeletal lesions which would support the idea that skeletal stress is evidence of frailty rather than resilience (Kyle et al. 2018). In their twenty-year review of the osteological paradox DeWitte and Stojanowski determined the osteological paradox while often cited was rarely a key aspect of modern bioarchaeological research (DeWitte and Stojanowski 2015). The authors suggested researchers address the osteological paradox directly and reiterate Wood and colleagues call for examination of samples from simple sites, contextualized perspectives, incorporating subadults into the analysis, associating stress markers with demographic phenomena, and digging deeper into the etiology of skeletal lesions (DeWitte and Stojanowski 2015).

This thesis was designed to incorporate the suggestions put forth by Wood and colleagues and emphasized by DeWitte and Stojanowski (2015) and Wright and Yoder (2003). For example, this work applies an intra-site approach to determine if there are differences in the bone histology between individuals from the well contextualized Kilkenny Union Workhouse population sample who exhibit osseous lesions and those who do not. The thesis incorporates

sub-adults into the sample and, while it does not examine lesion etiology, it considers the effect of lesion presence on the pattern of cellular remodeling in the rib histomorphometry.

2.5.4.3. The Third Wave of Biocultural Bioarchaeology

The third wave of biocultural engagement in anthropological research emphasized the responsibility of bioarchaeology to contribute to the understanding of the *lived* experiences of people in the past by incorporating biological, behavioral, ecological, and social research into bioarchaeological questions (Agarwal and Glencross 2011). In a sense, this wave encompasses the previous two waves while emphasizing the contextualization of the skeleton as a framework from which to build a *social* bioarchaeology. Social bioarchaeology recognizes the body as a multidimensional entity shaped by a myriad of biological and cultural factors. Through this molding, the body adapts to form what anthropologists refer to as human variation, the phenotypic traits observed and recognized on the body in life and in death that often have social, cultural, and biological consequences. These traits can also be expressed through evidence of health, disease, and identity in the skeleton and reflect the impact of age, sex, gender, culture, religion, and class (Dufour and Piperata 2018; Zuckerman, Kamnikar, and Mathena 2014).

Modern examples of research that have embraced a social biocultural approach to bioarchaeology have looked at the biological effects of stressors such as the Black Plague during the medieval period (AD 1347–1351) in London (DeWitte 2010; DeWitte and Wood 2008), the Great Famine (1845-1852) in Ireland (Geber 2015), and the Mississippi State Asylum in Jackson, Mississippi dating to the eighteenth and nineteenth century (Zuckerman, Kamnikar, and Mathena 2014). Biocultural methods are also employed to examine lifestyle and hardship in immigrant populations such as among the nineteenth century first generation European settlers to New Zealand and Australia (Buckley and Petchey 2018; Snoddy et al. 2020) as well as the impact Spanish settlers had on native populations like the Mórrope in the Central Andes (Klaus and Tam 2009). Contemporary migrants such as those immigrating from Mexico into Texas, Arizona, and California have begun to receive attention in forensic anthropological investigations with the primary aim of identification, with some studies assessing levels of stress markers and trauma as signs of motivation for migration (Beatrice and Soler 2016; Birkby, Fenton, and Anderson 2008; Spradley et al. 2019).

In these biocultural studies, pathological lesion analysis and other stress markers are situated within the cultural context of the sample with emphasis placed on the allocation of resources and the response to suffering such as how assistance from the government or healthy community may have affected the health outcomes of those who are more at greater risk of death (Tilley and Oxenham 2011; Vlok 2017; Casserly and Moore 2018; Smith et al. 2018). Elucidating the attitudes and actions of the government and community towards more vulnerable individuals in the population can help the researcher navigate the effects of stress and how cultural buffering mechanisms are functioning (Zuckerman and Martin 2016). For example, if those who are suffering from disease are cared for and included within the community, as people with leprosy are thought to have been in late medieval England (Roberts 2011), then their cultural buffering system is working in favor of the vulnerable to reduce the risk of death. On the other hand, if individuals with disease are stigmatized, their cultural buffer is not working to reduce their frailty and may be actively increasing the risk of death through reduced access to helpful resources like shelter, medical assistance, and nourishment (Zuckerman and Martin 2016).

2.6. CHAPTER SUMMARY

This chapter described the basics of bone biology and the histological variables necessary to understand the foundation of this research. This chapter also discussed some of the ways bone histology has been used in bioarchaeological studies, including how the incorporation of values from $\delta^{13}C$ and $\delta^{15}N$ light stable isotopes with bone histomorphometry can show whether the introduction of workhouse relief food in the form of maize helped improved bone health. Finally, this chapter explained the evolution of the biocultural approach over time and addressed two specific theoretical concepts that have grown from the approach that helped guide this research: the osteological paradox and structural violence. To address concerns regarding the osteological paradox, this thesis was conducted using an intra-site analysis of a well-contextualized, short-term use burial sample.

The following chapter will describe the period of the Great Famine and how historical contextualization of the conditions the poor lived under can situate this event because of structural violence. It will also introduce the bioarchaeology of the Great Famine and describe how pathological analysis of the skeletons and bone histology of the victims who died in the

workhouse is essential for illuminating their life experience and others who suffered in poverty throughout nineteenth century Ireland.

CHAPTER 3: STRUCTURAL VIOLENCE IN NINETEENTH CENTURY IRELAND

Contributions to a socially active bioarchaeology can be achieved through a number of lenses. Some researchers have actively worked to engage with local communities, taking care to consider their traditions and feelings about the study of the remains of their ancestors through public outreach, including them in the excavation work, and arranging ceremonies to honor the dead (Buckley and Petchey 2018). Some have investigated the social implications of biological sex and gender in motherhood and child rearing (Miller et al. 2020a) and to address the lack of representation of children in bioarchaeological research (Kamp 2001). Others have more directly addressed human rights issues and contributed to the greater socio-political conversation, becoming participants in social activism by looking into the consequences of colonialism, war, and poverty through the lens of violence theory (Alfsdotter 2019; Farmer, 2003; Galtung 1969; Harrod and Martin 2014; Nystrom 2014). This thesis seeks to understand how homelessness, malnutrition, and disease, affected the body on a cellular level in nineteenth century Ireland. However, it is also important to understand that these biological consequences are the product of a hierarchy of vulnerability. This section describes how those who ended up in the workhouse, the most frail of the population, were victims of a structurally violent society that revealed itself amid the tragedy of the Great Famine.

3.1. STRUCTURAL VIOLENCE

The term "violence" in popular understanding is often associated with acts of direct interpersonal harm where weapons or the physical body are used to intentionally inflict pain, suffering, or death on another individual; but violence can be both indirect and direct in nature. Galtung explained violence as a form of harm that widens the gap between an individual's "actual somatic and mental realizations" and their "potential realizations" (Galtung 1969: p. 168). In his 1969 paper, "Violence, Peace, and Peace Research", published in the *Journal for Peace Research*, he identifies the many forms that violence can take, emphasizing that the absence of a violent actor does not indicate the absence of violence. In cases where an actor can be identified, Galtung considers the violence direct while in cases where there is no specific actor the violence is considered structural or indirect violence. In other words, structural violence can occur any time a system prevents individuals, groups, or societies, from reaching

their social, economic, and biological potential (Galtung 1993). The violence is "structural" because it exists within a socially constructed environment and it is "violent" because it leads to physical or psychological harm or death (Farmer et al. 2006). To quote Galtung, "…if people are starving, when this is objectively avoidable, then violence is committed, regardless of whether there is a clear subject-action-object relation…" (Galtung 1969: p. 171).

Structural violence is often considered an invisible form of violence because it is embedded in long-standing and multi-generational repetitious structures (Farmer 2004); though some argue that it is the repetition of structural violence that perpetuates its invisibility (Winter 2012) and Galtung, while stating that structural violence is silent, also says that it can manifest "as natural as the air around us" (Galtung 1969: p. 173). Direct violence is more obvious and fluctuates over time whereas structural violence is more static and can cause greater long-term harm. Given the original definition by Galtung, structural violence applied to bioarchaeological studies is what researchers are seeing first-hand through the archaeological record when they perceive an individual skeleton as not having met it is biological potential in some way or when death and disease have occurred because of the restriction of resources by those on the upper end of the social hierarchy (Klaus 2013; McDade 2008).

3.1.1 BIOARCHAEOLOGY OF STRUCTURAL VIOLENCE

Paul Farmer (1959-), a notable medical anthropologist, brought the theory of structural violence into the anthropological sphere with his research on the ongoing AIDS epidemic in Haiti. Farmer, along with his colleagues working in Latin America, worked to understand how systems of oppression cause long-term harm in "underdeveloped" regions of the world. Farmer considers the effects of neoliberalism as a major agent in the harm caused by structural violence, a viewpoint that clashes with the Western liberal perception of self (Farmer 2004). For Farmer, "structural violence is violence exerted systematically—that is, indirectly—by everyone who belongs to that order" (Farmer 2004: p. 307).

The embrace of nuance inherent in the study of violence was well understood in cultural anthropology at the turn of the century, and it was not long before bioarchaeologists began to understand how to contextualize more far-reaching notions of violence into the study of people from the past, pushing bioarchaeological research into the third wave of biocultural research

(Martin, Harrod, and Pérez 2012). Since bioarchaeologists are keenly aware of the impact of social constructions such as gender, race, and class on health, they are also poised to answer questions about the impact of inequality in the context of violent societies (Larsen 2018). Violence studies in bioarchaeology can be focused on the origin and evolution of violent behavior (is human nature violent?), the rituals of violence (how violence is committed), and the culture of violence (who commits violence and who is impacted by it) (Stone 2012; Martin and Harrod 2015). However, most of these studies are focused on direct violence and how it is observed in the archaeological record.

Due to the narrow understanding of violence in bioarchaeological research, Klaus recommended a broadening of the concept of violence to include theories discussed in social and medical research throughout the twentieth century (Klaus 2012). His case study example of health in colonial Peru marks the beginning of a new era of violence research in bioarchaeology that specifically focuses on interpreting invisible (indirect) trauma that is arguably more harmful than direct violence through a heavily contextualized bioarchaeological lens (Klaus 2008; Klaus and Tam 2009). This can be done through the observation of features in bone that are indicative of embedded structural oppression with long-term consequences for the afflicted groups (Schrader 2019).

Evidence of structural violence through skeletal trauma and pathological changes has been observed in medical practices (de la Cova 2011; Nystrom 2014), from a feminist perspective (Atwell 2017; Stone 2016), throughout periods of colonization (Klaus and Tam 2009; O'Donnabhain and Lozada 2014; Redfern et al. 2019; Schrader 2019), at massacre sites (Alfsdotter 2019), and in immigrant populations (Pearlstein 2015; Redfern et al. 2019). Research by Martin and colleagues, for example, focused on the impact of a post-reconstruction lifestyle on the health of a group of Afro-Americans from a historic cemetery in Mississippi (Martin and Armelagos 1985). This group, who were recently emancipated yet bound by systemic constraints such as unequal access to healthcare suffered great inequities that manifested in their skeletal tissue as lesions indicative of metabolic and infectious disease. In histological analyses, the ribs were porous with high rates of resorption compared to formation which indicated an imbalance in bone remodeling, or a disruption in bone formation homeostasis. This Afro-American skeletal sample was seen as more stressed than their well-

nourished groups and the previously enslaved groups. This was explained by historical records showing a lack of access to nutritious food resources to Black Americans in the post-reconstruction South. While Martin and colleagues did not specifically contextualize their observations within a theory of structural violence in this paper, the study was an early example of how, in this case, structural violence can be observed in the archaeological record and an early glimpse into Martin's later work on the manifestations of violence (Harrod and Martin 2014; Martin, Harrod, and Pérez 2012; Martin and Tegtmeyer 2017).

3.2. POVERTY, DISCRIMINATION AND THE PENAL LAWS: EXPRESSIONS OF STRUCTURAL VIOLENCE IN NINETEENTH CENTURY IRELAND

In the nineteenth century, Ireland was a constitutional part of the United Kingdom through the Act of Union of 1801. While free trade and development capital for Ireland might have been beneficial outcomes, and many Irish people were initially in favor of the Union Act, the years that followed this agreement exemplify the complex sociopolitical and cultural relationship between the two countries that has existed for centuries (de Beaumont 2006; Campbell 2014).

When the Act of Union was enacted, Catholics in Ireland had been fighting for emancipation for more than a century (Sexton 2012). Prior to the Reformation, Ireland was still mainly under the control of Gaelic chieftains and regionally established Anglo-Irish lords who were established in Ireland for many years and, in many ways, had become accustomed to and even identified with Irish culture, including the dominant Catholic religion. However, when Henry VIII broke from the Catholic Church, he established the Protestant Church of England and associated Church of Ireland and enacted the Act of Supremacy, which declared him the "Supreme Head" of both churches. With a combined church and state agenda, Henry VIII did not just aim to rule over Ireland, but he intended to change Ireland into a Protestant nation. Though many Anglo-Irish lords opposed the Reformation and rebelled under the leadership of Earl Thomas Fitzgerald in 1534 and with support from other Catholic Nations including the Holy Roman Emperor Charles V and the Pope, the Earl was ultimately defeated (Campbell 2014). The struggle for religious freedom for Catholics in Ireland intensified in the centuries to come. For Catholic Ireland, emancipation meant repealing all the Penal Laws, a series of restrictions enacted from 1695 in retaliation for the Irish support of the Stuarts and James VI against William of Orange in England (Beckett 1981; Connolly 2000). The Penal Laws were

considered the ultimate catalyst for the deterioration of Catholic society under direct British rule, especially since one of the most detrimental aspects of the laws was that they prevented Catholics from maintaining a position in the Westminster Parliament.

The Penal Laws removed nearly every right of a Catholic (Smyth 2012). They could not vote or practice their faith and Catholic children could not attend school nor go abroad for educational purposes. The right to purchase land was suspended and sons who converted to Protestantism were rewarded with the father's property after his death, leaving any remaining sons without an inheritance (Smyth 2012). Effectively, any means by which a person could gain wealth or improve their socioeconomic status was prohibited for the Catholics under the Penal Laws. For the next two hundred years, the Catholic emancipation campaign fought to retain their rights, culture, and independence.

3.2.1. POVERTY AND SOCIAL CONDITIONS

In Ireland (Figure 3.1.), the issue of religious discrimination was also an issue of class (Geber 2015). The lack of Parliamentary representation combined with other discriminations led to severe poverty throughout most of Ireland. Much of this poverty was due to the issue of land ownership throughout the country. In Ireland, large swathes of land were owned by absentee landlords (Smith 1993). Not only were many absent landlords either unaware or unconcerned with the conditions of the property, but in these cases, money from the rents paid was spent outside of Ireland (Woodham-Smith 1962), further blow to the minuscule economy. Other landlords were middlemen who bought land, divided the land into small portions, and rented these portions for large profits. Tenants had no rights on the property they rented and little to no security. Even if they held a formal lease, tenants could be ejected from the land at the will of the property owner for any reason.

Just prior to the onset of the Famine, the British Government instituted the Devon Commission a committee overseen by William Courtenay (1777–1859), the 10th Earl of Devon, instructed to report on the state of land occupation in 1843. Their findings, collected by a group composed mostly of landlords, were published in a detailed report in 1847. The Commission found that "the principal cause of Irish misery was the bad relations between landlord and tenant" (The Devon Commission 1845). The report dictated the state of the land on which the tenant lived

as very poor and noted that in many cases the family's only source of food was the potato, their only source of drink is water, and furniture, even a bed, is a rare luxury (Devon Commission 1845). Based on these descriptions, it is not difficult to imagine the suffering the poor would have experienced during the approximately thirty separate periods of food shortage that are known to have occurred throughout Ireland in the hundred years preceding the Great Famine. In 1790, for example, a two-year famine period wiped out 25% of the population in the province of Munster (Smyth 2012), and more than fourteen partial and complete famines struck Ireland in the decades between 1814-1845, just before the blight arrived. While this is another extreme example of famine, the poor regularly dealt with minor seasonal famines for months at a time.

A noticeable increase in the population of Ireland began in the mid-1700s when there were an estimated four million people in Ireland until 1841 when the census recorded 8.16 million in the country (though many economic historians believe the number was closer to 8.75 million or even higher) (Smyth 2012). While a similar population incline in England and Wales could be attributed to industrialization, industry in Ireland could only account for a small portion of the growth (Mokyr and Gráda 1982). In fact, counties with major cities such as Dublin and Belfast experienced population decreases. Surprisingly, the poorest districts across Ireland, particularly in the west, experienced the greatest increase in population during this time (Smyth 2012). While not as destitute as the west, the County of Kilkenny also witnessed an increase in residents, up to 200-300%. The small-pox vaccine, the growing textile industry, and cultural customs such as partible inheritance all contributed to the rise in the population in rural western regions throughout Ireland, but it was the versatility of the potato that drove massive change (Smyth 2012).

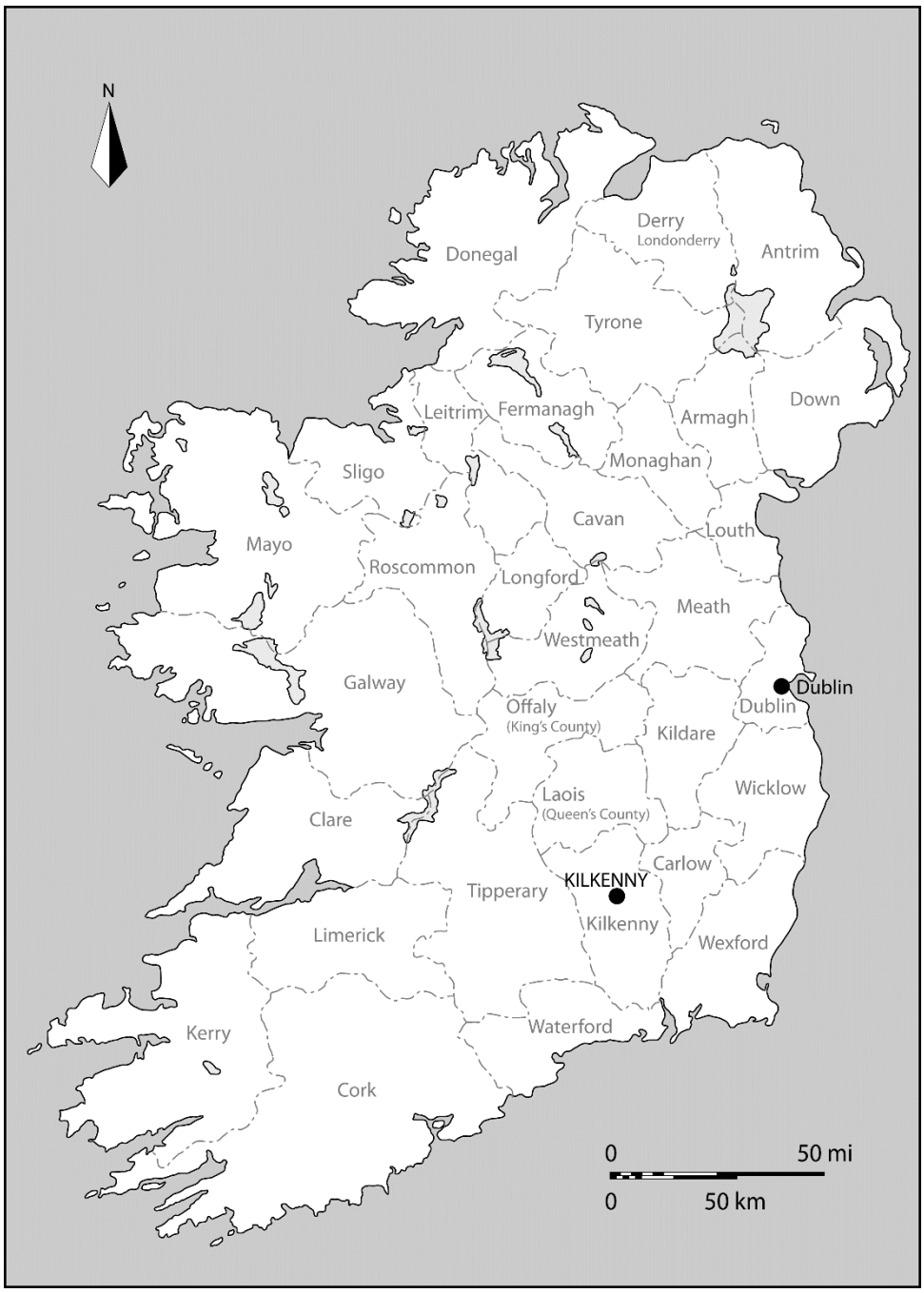


Figure 3.1. Map of Ireland. Template by Alan O'Rourke and adapted by Geber, 2015

Cheap, hearty, steadfast, and low maintenance, the potato rapidly became the crop of choice of the cottier, laborer, and small farmer in the decades before the Famine (Feehan 2012). However, as the population increased, in part due to the nourishment and relative reliability of the potato crop, and the Irish were dealing with the loss of their rights and the loss of their land, they became entrenched in a harrowing chicken and egg situation—while the potato was a lifesaver

for the poor, some of its redeeming qualities became consequential for those who subsisted off of little else (Geber and O'Donnabhain 2020).

Multiple varieties of potato were cultivated in the fertile soils of Ireland, but the Black Potato, the Apple, the Cup, and the Lumper varieties were ideal for their storing qualities and reliable seasonal returns (Feehan 2012). These potatoes could also thrive in harsh weather conditions and did not need much land to produce a bountiful harvest. The Lumper, specifically, became the primary crop of the farmer in the 1840s because it could produce four times as much food than any other crop per acre (Feehan 2012). This meant that landlords could subdivide smaller sections of land and rent to more people who would be able to survive thanks to the versatility of the potato. These bits of land, however, were either too small or not suitable to grow much else, leaving the tenants vulnerable to the occasional famine due to bad weather or a bad harvest. The consequences of a bad harvest were particularly devastating if the seed was also compromised, putting the following year's harvest in jeopardy (Feehan 2012).

Even if the potato season was productive, the poor still had to endure what were referred to as the "waiting months", the time between the exhaustion of the potato stock and when the next crop was ready to be pulled from the earth (Sexton 2012). The waiting months were usually July and August but sometimes could last as long as September or November. Though there were some potatoes that would come to market in the summer, such as the Kidney potato, these and other crops were often too expensive for the poor (Sexton 2012). The study by Geber and colleagues, which used microparticle and proteomic analyses to examine diet through the contents of dental calculus, showed that while the potato was the main source of food, the poor in Ireland also took advantage of other foodstuffs such as eggs and wheat whenever they could, which was not often (Geber et al. 2019).

Occasionally, relief food would be sent for support until the new crop matured. Humphrey O'Sullivan, an Irishman writing about his travels in the mid-1800s, reported only oatmeal and maize were keeping the poor from "stark famine" in the town of Callan in County Kilkenny (only 20 kms south of Kilkenny City) during these waiting months (O'Sullivan, 1979). Meanwhile, the Irish were exporting homegrown grains to England and abroad on the scale of 460,000 metric tons a year. Some believe the export of Irish crops contributed to the onset of

famine since many farmers were forced to export their crop to pay rent. It is thought that Ireland was producing enough food that if the harvest went to local markets or be consumed by the farmers that the growing population would be satisfied (Donnelly 1996; Smyth 2012).

3.2.2. THE POOR LAWS AND THE WORKHOUSES

The state of Ireland in the mid-nineteenth century was complex. For centuries Ireland had been living under varying levels of direct and indirect English rule that benefited England and the wealthy predominantly Protestant elite in Ireland (Kinealy 2002). On the one hand, this meant relaxed trade agreements between Britain and Ireland; on the other hand, a high price was paid with the establishment of the Penal Laws which made 80% of the Irish population vulnerable through a lack of Parliamentary representation, insecure housing, and a dependence on a single crop (Clarkson and Crawford 2001). So that when the potato blight arrived, no matter the efforts of Parliament to assist it, Ireland was primed for catastrophe.

While they had been slowly gaining more freedoms, the push for Catholic emancipation from the Penal Laws gained momentum just a decade prior to the Great Famine with the Catholic Relief Act 1829 (Campbell 2014). Catholics in Ireland were finally relieved of the Penal Laws and allowed a position in the Westminster Parliament. While sitting in the House of Commons an Irish barrister turned politician named Daniel O'Connell, who was referred to by the Irish as "The Liberator" for his rigorous activism in favor of Catholic rights, begrudgingly voted for a new law for a more effectual relief of the destitute poor in Ireland—the Irish Poor Law Act of 1838 (O'Connor 1995). This act was based on the Poor Law Amendment Act for England and Wales that incentivized the lowest classes to earn their way out of poverty, an attempt to relieve the poor of their own lethargy. To use an American phrase, they needed to "pull themselves up by their bootstraps".

The lives of the poor were now at the hands of the Poor Law Unions who did not have the resources to provide for the thousands of starving people across Ireland. Of the many responsibilities of the Poor Law Unions, the most controversial and difficult was the maintenance and control of the workhouses, one of the few sources of indoor relief that were available during the Famine. As a part of the Poor Law incentives, workhouses were constructed in each district where the "deserving poor" could work to receive food and accommodation.

These restrictions were enforced by the Board of Guardians, an amalgamate of high standing and prominent local people including clergymen and landowners who would determine who was allowed entry into the workhouse according to the Poor Law regulations.

According to the 1838 Poor Law Act, those who were deserving were "such destitute poor Persons as by reason of old age, infirmary, or defect may be unable to support themselves, and destitute children; and in the second place, such other persons as the said Guardians shall deem to be destitute poor, and unable to support themselves by their own industry, or by other lawful means" (Irish Poor Law Act 1838). Though O'Connell did not agree that the Poor Law was enough to help Ireland and thought that Ireland could not prosper unless it had a legislature of its own, he understood that something must be done to lessen the distress of his country. Regarding his vote on the Poor Law, O'Connell stated that the law "will not succeed in mitigating the evils to be found in the present state of Ireland … I yield to the necessity, while I regret it" (O'Connell 1837, p.1).

In 1849 it was reported that 932,284 women, men, and children of all ages were housed in the workhouses in use across Ireland (Luddy 1995). Once inside, the inmates were segregated based on sex and age and those who were older than two years old were separated from their mothers (Geber 2015). Since the goal of the workhouses was to entice the poor to make a better life for themselves, workhouses were designed to be undesirable places to live; they were damp and cold with few windows (O'Connor 2005). Once they achieved admittance, inmates were required to participate in harsh labor including working the corn mills or spinning wheels in exchange for food and shelter, but were only supplemented with basic dietary requirements, usually in the form of bread, milk, and maize (Crawford 1984; Geber et al. 2019; Thomas 2012). Poor nutrition, overcrowding, and unsanitary conditions caused the rapid spread of disease and increased rates of mortality in the workhouses and the associated fever hospitals, especially as the Famine became more widespread and more people became dependent on the workhouse for shelter and food relief.

3.3. AN GORTA MÓR: THE GREAT HUNGER (1845-1852)

In 1841, the population of Ireland was a robust 8.2 million, but the socioeconomic disparity was glaring, particularly between the urban areas of the east and the rural regions of the west

(Smyth 2012). According to the census that year, three million people were considered landless agricultural laborers and over one million were cottiers who leased small parcels of land barely big enough to grow their potato crop. While it was possible to live a healthy life under those circumstances, the census also recorded that 40% of people in Ireland were living in an estimated 500,000 bothán (Census of Ireland Commission, 1843; Clarkson and Crawford 2001; Geber and O'Donnabhain 2020). Bothán were one-room mud huts considered fourth-class accommodations "unfit for human habitation" (The Devon Commission 1845). Often, multiple families would reside in one hut and share the product of a single piece of land. When rents could not be paid it was not unusual for these structures to be flattened, leaving all its occupants homeless. Near the end of the Famine, the 1851 census recorded a shocking statistic, the number of recorded bothán in the country was reduced from half a million to 135,000 (Census of Ireland Commission 1853). What is evident from the 1841 and 1851 census data regarding class and housing is that nearly half of Ireland was living in or on the verge of poverty merely four years before the Famine devastated the country.

While the beginning of the Great Famine is widely recognized from the year 1845, the devastation did not occur in one fell swoop. At first, it appeared there would be a plentiful harvest that year and in July, the potato crop in Ireland was described by the *Freeman's Journal* as, "never before so large and at the same time so abundant" (Woodham-Smith 1962: p. 38). Even though that crop soon turned black from the blight, the British Government treated it as an isolated episode from which Ireland could recover as they had from numerous other famine episodes that occurred in the early to mid-1800s. While it is true that Ireland did often bounce back from previous intermittent bouts of famine that plagued the century as evident from the increase in the population at the time, the dependence on the potato made the poor especially vulnerable to food shortages. In the case of the blight of 1845, there was an exceptionally and unexpectedly long-term food shortage (Kinealy 2002).

The total loss of a crop occurred for a number of reasons. A bad harvest can be the result of a months-long freeze, flooding, pests, parasites, or pathogens (Feehan 2012). In the case of an attack from disease, crops are more susceptible if they have not developed resistance through genetic variability. The Lumper, while easy to maintain and generous in its yield, is one such genetically homogenous modern crop that, without preventative husbandries such as crop

rotation and hygienic storage, would succumb quickly to disease (Feehan 2012). The first reports of rotted potatoes were written in August of 1845. The potatoes were described as smelling dreadful, like the scent of death, the stems and leaves were blackened, and the tubers rotted through. The culprit, a fungus known as *Phytophthora Infestans*, had originated in North America and spread rapidly to Europe—first into Belgium in June and then the Netherlands, France, France, Germany, England, and eventually Scotland and Ireland (Campbell 2014). Almost as soon as it arrived the blight destroyed nearly all of Ireland's potato crop and had the most devastating effect on the Lumper, the primary means of sustenance for the poor in Ireland (Kinealy 2002). The blight would cause the potato to turn completely black even while the stalks remained seemingly healthy. In some cases, desperate people would attempt to boil the rot out of the potatoes to make them edible, but they would still cause sickness. Since the poor rarely cultivated alternative crops, they had neither a primary food substitute nor the capital to purchase food from the local market, which was rising in price due to the potato shortage (Feehan 2012; Woodham-Smith 1962).

3.3.1. THE BRITISH GOVERNMENT'S RESPONSE TO THE FAMINE

At the onset of the Famine in 1845, Sir Robert Peel (1788-1850), on his second term as Prime Minister and leader of the Conservative Party with many years of experience in serving as Chief Secretary in Ireland, disrupted Parliament by repealing the Corn Laws. The Corn Laws taxed imported grain, rice, and other foods from the colonies and their repeal was a deeply controversial move that divided the House and turned the Famine into a party issue. However, if Ireland was to be saved, the free import of relief food was necessary. Unfortunately, it was difficult for Peel to gain Cabinet support for the repeal as there were many who believed that the Famine was over-exaggerated, a hoax designed by Irish agitators. After the motion to repeal the Corn Laws failed twice, Peel resigned, only to be reinstated by Queen Victoria after a suitable replacement could not be found. Disregarding the opinion and lack of support from his own party and at exuberant cost, Peel bypassed the Treasury and ordered a substantial amount of maize from North America to sell at a cheap cost in Ireland, a move that likely extended the lives of many in 1845-46 (Gray 2012; Kinealy 2002), but aggravated the laissez-faire style of market liberalism appreciated by the English. Many believe this style of governing—the belief that the government should not interfere in private enterprise or engage in the market as a

buyer—is what kept the poor from receiving the assistance they needed to survive the Great Famine (Ó'Gráda 1995). Of course, revisionist researchers argue that it was Peel's macroeconomic policies that drained the Treasury and forced the Whigs, who came to power after Peel resigned as Prime Minister in 1846, to minimize relief efforts in Ireland (Read 2016).

Alternative plans for relief implemented by Peel included the formation of local committees run by Relief Commissioners to raise money for those in need. Under the commission, much of the responsibility was placed on the landowners to provide employment to those who resided on their estate. Another relief measure, The Irish Board of Works, was designed for the poor to receive a small wage in exchange for performing physical labor, such as building roads. This form of outdoor relief was barely enough to feed a family and sometimes would not be paid out for days or weeks after the work. The third component of Peel's relief plans involved the construction of fever hospitals to prepare for the inevitable rash of fever that always followed episodes of famine (Gray 2012).

When the money for public works ran out and the initiative failed to provide much relief, the government passed the Temporary Relief Act 1847, which helped relieve the poor of the devastating resurgence of the blight in the previous year through the establishment of soup kitchens that served "stirabout", a porridge containing some combination of maize, rice, and oats (Gray 2012). While this measure likely saved many lives, it only lasted about half the year, from February to September before the British Treasury, under the control of Charles Trevelyan (1807-1886), the Assistant Secretary who was effectively in charge of Irish relief efforts, determined the landed gentry in Ireland should find ways to provide for the poor without government intervention (Gray 2012).

Trevelyan, who was knighted in 1848 by Queen Victoria in recognition for his contributions to the "relief of the poor in Ireland", believed that inherent Irish moral failure was the cause of the suffering during the Famine. In *The Irish Crisis* (1848), Trevelyan acknowledges the danger of depending on a single food source and admits that the consequences of this type of diet had "long been foreseen by thinking persons" (Trevelyan 1848: p. 2) and while numerous calls to action were requested by the Commissioners of Parliament residing in Ireland after the abandonment of the Temporary Relief Act, Trevelyan repeatedly denied them (Gray 2012).

Eventually, instead of assisting the poor through government funds, the Poor Law Extension Act was filed so that poor relief could be provided to the destitute but only if they leased less than a quarter acre of land (Crawford 1984; Gray 2012). This initiative, perhaps inadvertently, led to the evictions of many small landholders, leaving them with no place else to go but the workhouses.

3.4. MASS BURIALS AT THE KILKENNY UNION WORKHOUSE

In 2005, a burial site was discovered on the former Kilkenny Union Workhouse grounds (Figure 3.2) during a development project (Figure 3.3). An archaeological excavation was conducted the following year by Margaret Gowen and Co. that uncovered 63 sub rectangular pits arranged in rows that were one meter apart (O'Meara 2010). Together, the pits contained the remains of at least 970 people including 425 adults and 545 children interred in simple pine coffins and layered on top of one another (O'Meara 2010; Geber 2015). Consequential bioarchaeological analysis of the human remains by Geber and colleagues has focused on the paleopathological evidence of disease, dietary isotopes, and microparticle analysis with the aim to elucidate the life experience of the poor prior to and during the Famine (Beaumont et al. 2013; Beaumont and Montgomery 2016; Geber 2015; Geber and Murphy 2012; Geber et al. 2019; Geber and O'Donnabhain 2020). This research has discovered a plethora of evidence to suggest the poor suffered from severe malnutrition, metabolic and infectious disease, and likely psychosocial stress resulting from their impoverished status (Geber 2015).

The Kilkenny Union Workhouse sat on what was then the outer edge of Kilkenny City, an old, well preserved medieval city in the province of Leinster (Geber 2015). It was one of the largest institutions of its kind in Ireland with four accommodation blocks and sat on a total of four hectares of property. Initially the workhouse was built for the capacity of 1,300 inmates but during the height of the Great Famine it provided relief for over 4,300 hungry, poor, and sick people, nearly four times its intended capacity (Geber 2012). Historical records from local newspapers and written records from the workhouse minute books recorded the Kilkenny Union Workhouse population to contain at least half adults and half children, many of whom would have entered the workhouse as orphans (Geber 2012). The demographics from the mass burials mimic the minute book records. Nearly half of the skeletons recovered from the burials are

those of children while the adults are split almost evenly between those with biological sex estimated as male and female.

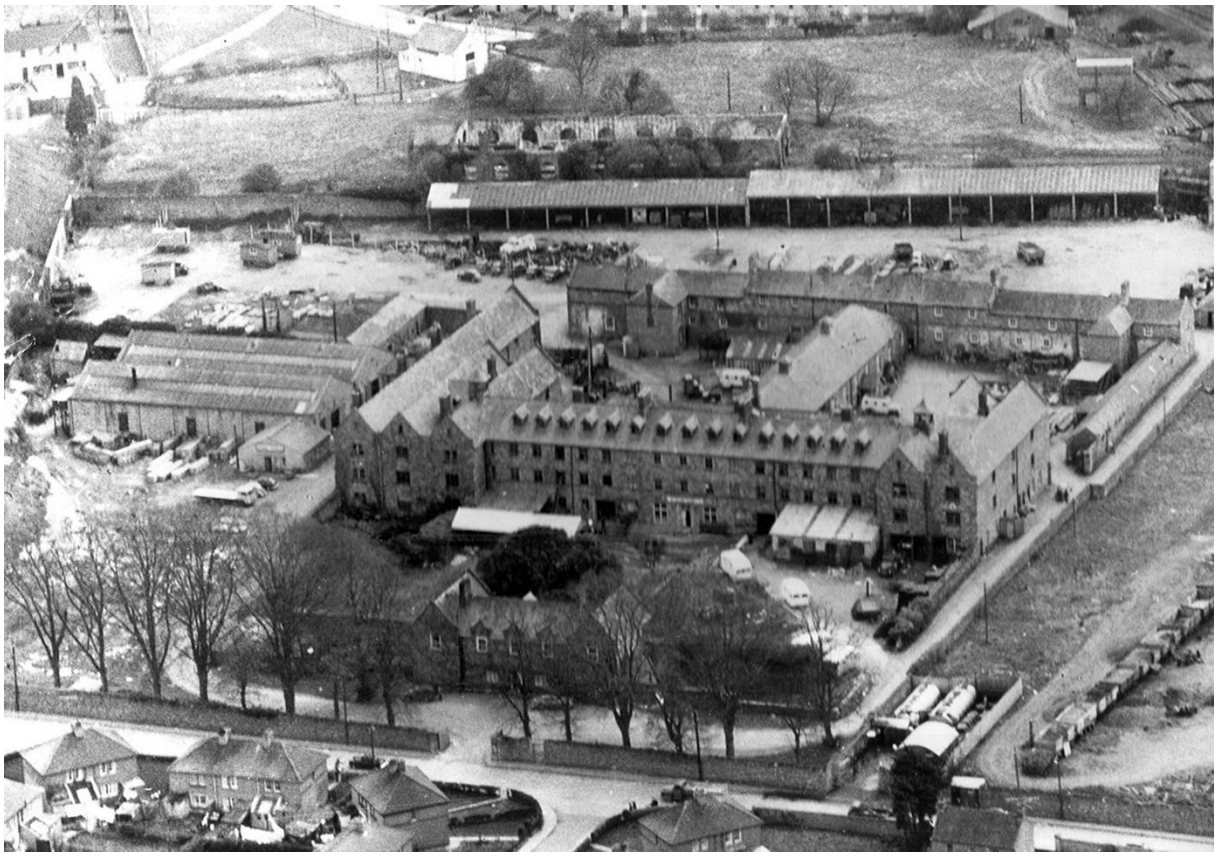


Figure 3.2. Aerial photo of the Kilkenny Union Workhouse. Photo courtesy of Karyn Deegan via the Kilkenny County Library

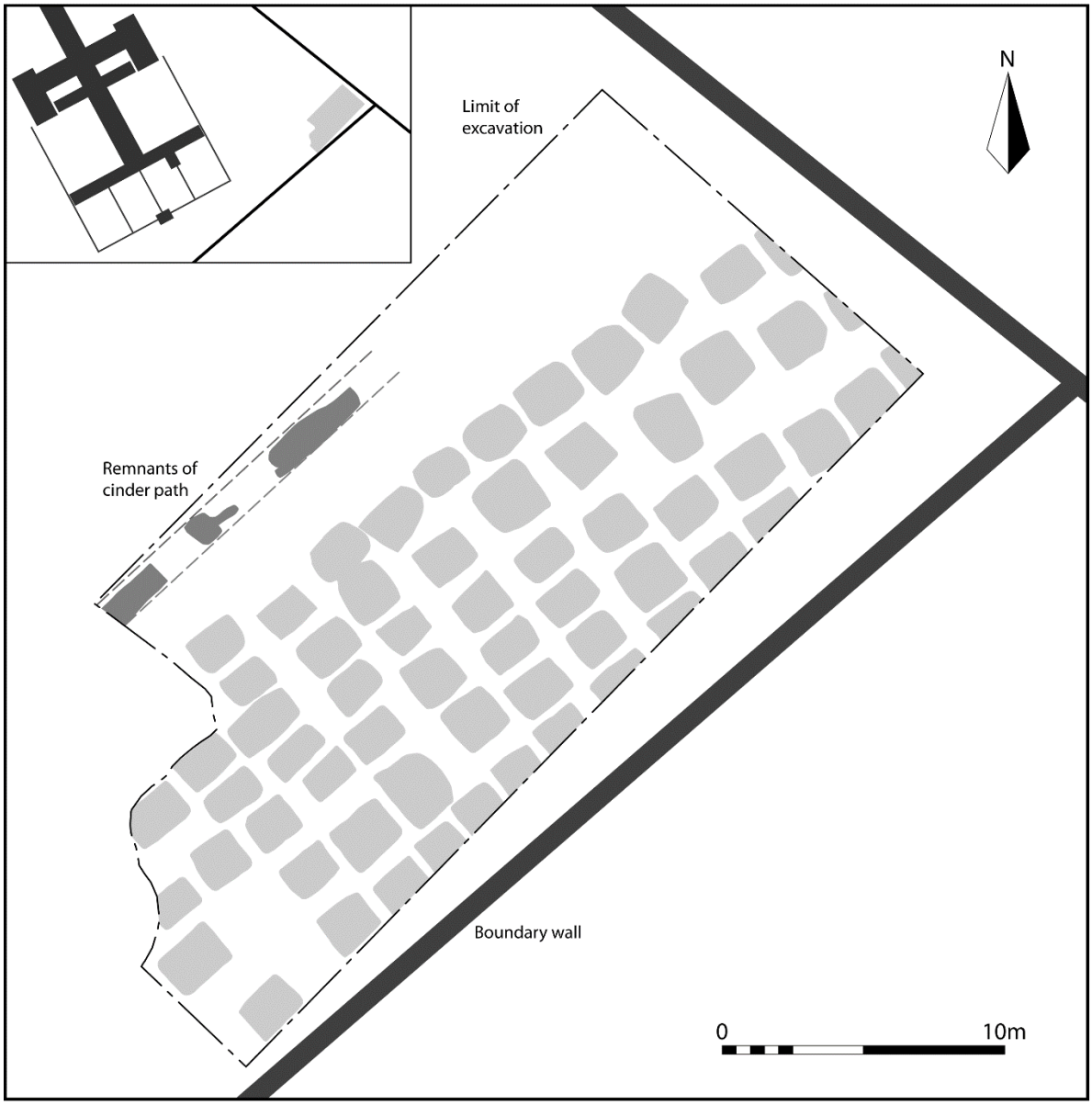


Figure 3.3. Illustration of mass burials (in light grey) on the Kilkenny Union Workhouse grounds. Source: Geber, 2015

Nineteenth century Kilkenny was highly stratified with abundant wealth and industry as well as abhorrent destitution. While Kilkenny, with its marble and textile industry was described as having the most fashionable shops in the country, it was also marked by mud dwellings occupied by multiple families that took up two thirds of the residencies in the city (Neely 1989; Geber 2015). Though the predominant language was Irish, it was mostly spoken by the lower

classes while the middle and upper classes spoke English. This would become an important factor that inhibited access to resources for those who needed it most during the Famine.

3.4.1. PALEOPATHOLOGICAL ANALYSIS OF THE SKELETONS AT THE KILKENNY UNION WORKHOUSE

There were a wide range of infectious and metabolic diseases that affected the poor in Ireland prior to and throughout the Famine which were recorded by the census and described by a number of accounts from doctors, workhouse officials, and travelers (de Beaumont 1839; Census for Ireland for the Year 1851; Nicholson 1851). Infectious diseases included "famine fever", dysentery, "famine diarrhea", cholera, smallpox, and tuberculosis; common metabolic diseases resulting from lack of nutrition included marasmus, dropsy ("hunger edema"), scurvy, and likely pellagra (though this was not a recognized disease at the time) (Crawford 1984).

In his book *Victims of Ireland's Great Famine*, Geber uses first-hand quotations from locals and travelers to the region who emphasize the ill health and bad living conditions of the poor in Kilkenny (Geber 2015). Often, they are described as living in filth, such as in a quotation by the local Kilkenny landlord, and former MP of the Irish Parliament prior to the Act of Union, William Tighe who mentioned the successive relapses of rheumatism and pleurisy inflicting the poor who slept on straw or damp clay. Geber goes on to explore the wretched state of the poor while Ireland was in Union with Great Britain by investigating the physiological experience of Famine through a social bioarchaeological lens and focusing on the gross pathological indicators of disease visible on the skeleton. These markers revealed trends in morbidity and mortality as well as patterns of labor and resource distribution between sex and age groups that show how the victims experienced the Famine from an individual and community perspective within the workhouse (Geber 2015).

While there is bias in the selection of individuals who were allowed into the workhouse and this sample is not representative of the entire population of the poor in Ireland in the nineteenth century, it does represent the life experience of the one million people who died during the Famine and the threat that loomed over the one million who fled the country. The following sections detail the types and severity of infectious and metabolic disease identified in the workhouse sample.

3.4.1.1. Evidence of infectious disease

Geber reported that tuberculosis, respiratory disease, osteomyelitis, and syphilis were present in 15.8% of the people who were discovered at the Kilkenny Union Workhouse. Individuals with evidence of infectious disease are underrepresented in the paleopathology of the workhouse sample compared to the reports of disease in census records (Geber 2015) and, as a result, are underrepresented in the subsample used for bone histological analysis as well. The impact of infectious disease on bone histology was discussed in Chapter 2, Section 2.5.

Of the nine-hundred and seventy individuals in the Kilkenny Union Workhouse sample, only seven presented evidence of tuberculosis. In one of those cases the lesions were inactive at the time of death. Tuberculosis mostly affected the spine and the ribs with osteolytic destruction of the vertebrae and new bone formation on the surface of the ribs. Other lesions attributed to tuberculosis were observed in the pelvis, the mandible, the hands, and the arms. In one nine-year-old child, a lesion was observed in the frontal bone just superior to the right orbit. Geber emphasizes the lack of representation of tuberculosis in this sample and suggests death occurred before the lesions had time to appear, especially since the strength of immune defense of the infected person is a crucial aspect of recovery in victims of tuberculosis (Geber 2015).

General respiratory disease was also prevalent in Ireland prior to and throughout the Famine, especially in those who lived in the botháns (Geber 2015). Respiratory illness was often chronic and long-lasting and can affect the maxillary sinuses in the form of sinusitis, inciting new bone formation in the maxillary sinus walls and can affect the ribs (Aufderheide and Rodríguez-Martin 1998). Osteomyelitis, an infection of the bone marrow and a non-specific indicator of disease, was present in the lower extremities of nine individuals in the Kilkenny sample. Osteomyelitis in the skeleton is identified by the presence of a cloaca, a build-up of reactive new bone formation, and a sequestrum, or destroyed bone tissue (Aufderheide and Rodríguez-Martin 1998). While non-specific, osteomyelitis may be a symptom of tuberculosis but it can also indicate the person was suffering from typhoid fever or even smallpox. However, in the case of typhoid fever, the lumbar spine, ribs, and tibiae may have also been affected (Veal 1939).

3.4.1.2. Evidence of metabolic disease

Only a few metabolic diseases are identifiable in the skeleton; however, 51.5% of the skeletons recovered from the Kilkenny Union Workhouse sample exhibited evidence of metabolic disease (Geber 2015). Scurvy, the most identified disease in the sample, is a nutritional deficiency caused by a lack of Vitamin C (<10 mg/day), or ascorbic acid, in the diet that reduces or suspends the rate of collagen deposition (Fain 2005; Levine and Morita 1985). This leads to weakened connective tissue, osteopenia, cortical bone thinning, and defective endochondral growth in the long bones of children (see Snoddy et al., 2018). In life, scurvy results in painful symptoms including gingivitis and hemorrhaging, fever, and weakness among many other debilitating issues (Mays 2014; Ortner and Ericksen 1997; Ortner 2011).

Potatoes are high in Vitamin C. A fresh, uncooked potato can contain as much as 30 mg of the vitamin per 100 g, but this amount reduces as the potato ages in storage or when it is cooked (Geber and Murphy 2012). When boiled, the potato only retains 15-10 mg of its Vitamin C, but the poor in Ireland were consuming enough potatoes every day to achieve the required amount of ascorbic acid so scurvy was not common when harvests were successful (Crawford 1984; Geber and Murphy 2012). There was a high prevalence of scurvy across age and sex groups within the Kilkenny Union Workhouse population, which is likely due to the complete lack of dietary diversity in adults, children, and infants alike. The disease was present with some degree of confidence in 52% of the skeletons assessed with 66-68% of children to adolescents diagnosed and 31-49% of adults diagnosed (Geber and Murphy 2012). Sex differences in the frequency of scurvy were observed and males were diagnosed 1.7 times more frequently than females in the overall adult population. Geber attributes these sex differences to biologically necessitated Vitamin C intake between males and females.

Geber identified scurvy more frequently and confidently through pathological changes in the subadult group than in the adults from the Kilkenny workhouse, a trend that is common in other bioarchaeological samples from Europe (Geber and Murphy 2012; Mays 2014). Most of the lesions observed by Geber are due to hemorrhaging that causes porous and hypertrophic bone. These lesions were present in the form of subperiosteal new bone formation on the alveolus of the mandible, the palatine process, around the foramen rotundum, and on the supraspinous

portion of the scapula. Porotic pitting appeared in the skull, particularly the orbits and the greater wing of the sphenoid, the posterior surface of the maxilla, the medial surface of the mandibular ramii, and the buccal surface of the mandible (Geber and Murphy 2012; Geber 2015). It is impossible to know how many people suffered from scurvy during the Famine, but skeletal analyses from Kilkenny imply it was incredibly high compared to other nineteenth century populations (22% compared to less than 7%) (Geber and Murphy 2012; Mays 2014). Mays suggests the lack of prevalence for scurvy reported in archaeological research may have to do with the variation or error in interpreting Ortner's criteria for diagnosis (Mays 2014). To account for the variation in lesion prevalence and type in the Kilkenny sample, Geber used a scoring system to identify those with definite scurvy (*N*=156), probable scurvy (*N*=205), possible scurvy (*N*=138), and without scurvy (*N*=465). For the purposes of this comparative research, those with signs of scurvy were lumped together and compared to those without evidence of scurvy (Geber 2015).

Skeletal lesions present in the Kilkenny Union Workhouse sample also point to Vitamin D, calcium, and phosphorous deficiencies leading to rickets and osteomalacia, the adult form of rickets. Vitamin D can be obtained through fish, eggs, and butter, while calcium and phosphorus are prevalent in common foods such as meat, nuts, as well as fish and eggs. A lack of Vitamin D in the diet can impair the capacity for calcium and phosphorus absorption in the body, thereby inhibiting their deposition in bone and hindering the mineralization process in bone modeling and remodeling (Brennan-Olsen et al., 2019).

When Vitamin D is not supplemented through the diet or through sun exposure, it is obtained from stores throughout the skeleton, causing decreased mineralization in new and remodeled bone. This can result in bowing of the long bones, flattening of the femoral metaphysis and coxa vara, and thickened long bones in children (Brickley, Mays, and Ives 2010; Schattmann et al. 2016). Increased porosity and fractures will present in the bones of adults with Vitamin D deficiency (Brickley, Mays, and Ives 2010). Often, children suffering from rickets have difficulty with mobility and experience hypocalcemic seizures (Ward et al. 2007). In the Kilkenny population, rickets was observed skeletally in twenty-two (2%) subadult skeletons, primarily in the youngest groups, and manifested as long bone deformities. Residual rickets was observed in twenty-four adult skeletons and one adult skeleton displayed evidence of

osteomalacia observed as fractures in the lateral margins of the scapula, in the pubic ramus, and in one rib (Geber 2015).

Porotic hyperostosis and cribra orbitalia, two non-specific childhood pathologies identified as porotic lesions in the skull and often associated with either iron deficiency anemia or Vitamin B12 deficiency, were also present in the Kilkenny sample (Brickley 2018; Geber 2015). Porotic hyperostosis in the form of healed lesions with porosity was observed in the adults while subadults displayed active medium to severe porotic lesions. Cribra orbitalia, which manifests as porosity in the orbits, was identified in 20% of the overall population, 25% in subadults and 14% in adults. Geber states that subadult prevalence of cribra orbitalia may be higher because iron is required in children experiencing growth spurts (Geber 2015) or it could be due to Vitamin B12 deficiency because of the lack of this vitamin's reserve in children who were either not breastfed or breastfed by starving mothers with no B12 reserves due to Famine conditions. Cribra orbitalia due to iron deficiency anemia or Vitamin B12 deficiency was differentiated from that caused by scurvy by the presence of scorbutic specific lesions such as porous new bone formation and porotic pitting of the greater wing of the sphenoid bone. When compared to other nineteenth century populations, the rate of cribra orbitalia is high, which may reflect the stress induced by the experience of the poor in Kilkenny (Geber 2015).

3.4.1.3. Evidence of non-specific stress

Non-specific indicators of stress were prevalent throughout the skeletons buried on the Kilkenny Union Workhouse grounds in the form of enamel defects and hypoplasias, Harris lines, and growth retardation. The presence of enamel hypoplasias, observed as lines or pits in the tooth enamel, is due to the disruption of the deposition of enamel matrix during tooth development (Hillson 2014). Since tooth development occurs prenatally and in young childhood, dental enamel defects are often interpreted as signs of systemic early childhood stress (Floyd 2007; Goodman and Rose 1990). Defects may occur due to infectious disease (Masterson et al. 2017), malnutrition (May, Goodman, and Meindl 1993; Zhou and Corruccini 1998), fluoride exposure and weaning stress (Moggi-Cecchi, Pacciani, and Pinto-Cisternas 1994). However, susceptibility to the development of enamel hypoplasias are also likely genetic, wherein children who live long enough to develop the defects may be more resilient

during periods of stress than those who do not present with lesions but would have had similar famine experiences (Wood et al. 1992).

Harris lines present radiographically as transverse lines of hyper-mineralization in the trabecular tissue of the long bone metaphyses in children and adults. Their presence is thought to indicate increased mineralization due to the disruption in the deposition of the cartilage building chondrocyte matrix and continued deposition of the bone building matrix secreted by osteoblasts (Goodman 1981), though this is still being questioned (Miszkiewicz 2015). While their cause whether due to stress or normal changes due to growth is debated, they are often taken into consideration as non-specific indicators of disease when examining skeletal samples from known stressed populations (Boucherie et al. 2017; Marshall 1968). Harris lines were observed in nearly all of the subadult skeletons from the workhouse burials. Geber concluded that children between the ages of 6 and 12 years appeared to have exhibited Harris lines because of normal growth spurts while older children did not appear to have gone through a similar number of growth spurts (Geber 2014). This may be due to bone remodeling, which does occur quickly in children, or may be related to a lack of growth because of stress due to the Famine. Geber also observed that growth stunting occurred for 76% of the subadult population between the ages of 6 months and 12 years. The youngest of this group (6-12 months) were determined to be much smaller relative to the older age groups, which may indicate low birth weight due to maternal malnutrition, a common occurrence in populations experiencing food deprivation and famine (Geber 2014; Lumey 1992; Stein et al. 1975).

The prevalence of non-specific indicators of disease in form of enamel defects and hypoplasias, Harris lines, and growth retardation suggests that even when osseous lesions are not present in the skeleton, early childhood stress is apparent and likely occurred prior to as well as during the famine. This is particularly evident in the frequency of enamel hypoplasias in the adult sample and the small stature of the youngest individuals from this sample who probably suffered from malnutrition alongside their mothers. Both enamel hypoplasias and Harris lines are thought to be representative of periods of stress that disrupt the homeostatic rate of mineral deposition in teeth and bones. The presence of hyper-mineralized lines is also argued to be a non-specific indicator of stress in the dental cementum of teeth (Cipriano 2002) and in double zonal osteons,

though these interpretations are debated (Raguin and Drapeau 2020; Reichert and Mulhern 2018).

3.5. CHAPTER SUMMARY

Chapter 3 described the background to the inevitable failure of the potato crop that caused a national famine leading to the loss of nearly a quarter of the Irish population due to death and emigration. The reaction of the British Government to the crisis was discussed, which involved the implementation of poor relief in the form of maize at local workhouses. The next chapter describes the materials and methods used to interpret the effect of disease and malnutrition because of structural violence on the bone microstructure of those who died on the grounds of the Kilkenny Union Workhouse.

Table 3.1. Disease types and lesions in the Kilkenny Union Workhouse population identified and described by Geber (2015)

Metabolic Disease	**Osteopenia**	Low bone mass identified using radiographs and indicative of an imbalance in bone remodeling.
	Rickets/ Osteomalacia	Vitamin D deficiency diseases identified by bending of the long bone in children and combined porosity and fractures in the scapulae of some adults
	Scurvy	A Vitamin C deficiency disease identified as porosity in the greater wings of the sphenoid, in the posterior surface of the maxilla, in the medial surface of the mandibular ramii, on the buccal surface of the mandible, and on the anterior femur. New bone formation was also observed on the alveolus of the mandible, on the palatine process, on the supraspinous area of the scapula, and on the foramen rotundum.
	Rib Lesions	Identified as new bone formation on the ribs. These lesions may be non-specific indicators of metabolic disease but may also indicate infectious disease when paired with sinusitis and lesions indicative of tuberculosis.
Infectious Disease	**Tuberculosis**	A bacterial infection identified through combined observations of rib lesions with diffuse new bone formation or osteolytic abscesses, ossified plaques of lung tissue, osteomyelitis, spinal deformities and osteolytic destruction, subchondral destruction of the acetabulum and femoral head, and a cranial crater was present in one adult. Multiple elements also exhibited new bone formation including the public bone and ribs.
	Osteomyelitis	An infection of the bone marrow identified by the presence of a cloaca, a involucrum, and a sequestrum in the extremities of adults and children. May be indicative of tuberculosis, typhoid, smallpox, or other infectious diseases
	Sinusitis	An infection of the paranasal sinuses identified as new bone formation on the walls of the sinuses. May be indicative of pulmonary infection when rib lesions are also present.
	Syphilis	An infection caused by the *Treponema pallidum* bacteria that can affect the skeleton when it reaches the tertiary stage. Identified by a caries sicca lesions in the cranium of one adult that extended throughout the frontal bone and included the anterior portions of the parietal bones and the left eye socket. Hutchinson's incisors, mulberry molars, and gumma lesions were identified in the subadult sample.

CHAPTER 4: MATERIALS AND METHODS

The remains from the Kilkenny Union Workhouse burials were excavated by hand, cleaned with water, and then air dried. The bones were well preserved and in good physical condition for macroscopic pathological analysis. After the analyses were complete, the remains from the Kilkenny Union Workhouse were reburied in a crypt constructed adjacent to the site where they were discovered at the request of the public in Kilkenny City. The crypt is sealed, and the skeletons will remain there in perpetuity. To honor the victims who died on the grounds of the workhouse and to educate the public about the Great Famine, an audio-visual tour called *The Kilkenny Famine Experience* was launched in 2017 (Kilkenny Famine Experience, 2020).

The following chapter details the materials used in this sample and the methodology of procurement, preparation, and analysis of the ribs from the Kilkenny Union Workhouse population sample used in this research.

4.1. RIB SAMPLES FROM THE KILKENNY UNION WORKHOUSE

Prior to reburial, rib bones were sampled from 300 skeletons, which are the basis of this research. Analyses to determine age and sex were conducted by Geber (2015) using standardized methods. In the subadult population age was determined based on dental development and eruption (Broadbent et al. 1975), the examination of crown and root formation (Morrees et al 1963; Smith 1991; Liversidge 1998), and observations of epiphyseal fusion (Scheuer and Black 2000). In adults, age ranges were determined using standardized methods based on the sternal ends of the fourth rib (Işcan et al. 1984, 1985), the auricular surface (Lovejoy et al. 1985b), the auricular surface (Brooks and Suchey 1990), and cranial sutures (Meindl and Lovejoy 1985). A multi-factorial summary age determination technique (Lovejoy et al. 1985a) was used to generate an age range for each individual and a mid-value was assigned for each skeleton based on this range. In the adult population, sex was determined using standardized methods that include analysis of the features of the pelvis, cranium, and post-crania (Buikstra and Ubelaker 1994). The sample of ribs sectioned for histological analysis include 172 ribs from 168 adult individuals and 139 ribs from 127 subadult individuals (311 ribs; 300 individuals; 622 slides). Any rib that exhibited extensive periosteal lesions was not sampled and the final sample size was determined after scoring for preservation was performed.

Included in the final sample are 99 adults that make up 53.2% of the sample (55 females and 44 males) and 87 subadults that make up 46.8% of the sample (3 females, 3 males, 81 indeterminate sex) (Table 4.1). Both cohorts are representative of the overall sample excavated from the Kilkenny Union Workhouse grounds.

Table 4.1. Sex and age distribution (years) for the Kilkenny Union Workhouse sample included in this thesis after ribs with poor preservation were excluded

	Female			Male			Indeterminate			Total		
	n	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD
Adults	55	35.7	8.1	44	38.3	8.4	0	-	-	99	36.8	8.3
Subadults	3	15.2	.29	3	16.7	.58	81	8.4	4.4	87	8.4	4.4
Total	58	34.6	9.1	47	37	9.7	81	8.4	4.4	186	23.8	15.6

4.1.1. SAMPLE SELECTION

The ribs were prepared for histological analysis using undecalcified dry bone preparation techniques from modern, documented, and tested methods (Cho 2012; Cho and Stout 2003; Miszkiewicz and Mahoney 2016; Stout and Paine 1992). They were initially sampled at the National Museum of Ireland Collections Resource Centre in Swords, Ireland. For consistency with previous studies, samples were taken using a Dremel tool with a diamond blade saw from the midshaft of a middle rib, determined by measuring the shaft of the rib from the vertebral to the sternal end and cutting from one centimeter on either side of the halfway mark when whole ribs were available (Agnew and Stout 2012) (Figure 4.1). However, because the sample yielded variation in bone preservation, the "true" midshaft of the rib was not always identifiable as ribs were frequently fractured on one or both ends. Therefore, a one to two-inch section was cut from the shaft of each rib as close to the "true" middle as possible. Additionally, some of the ribs were lower ribs (9-12) or higher ribs (1-3) than the ideal middle-rib location (4-8) (Agnew et al. 2015; Crowder and Rosella 2007; Roberts and Chen 1972). To control for variation, only ribs that could be identified confidently as 3-9 were included (Crowder and Rosella 2007).

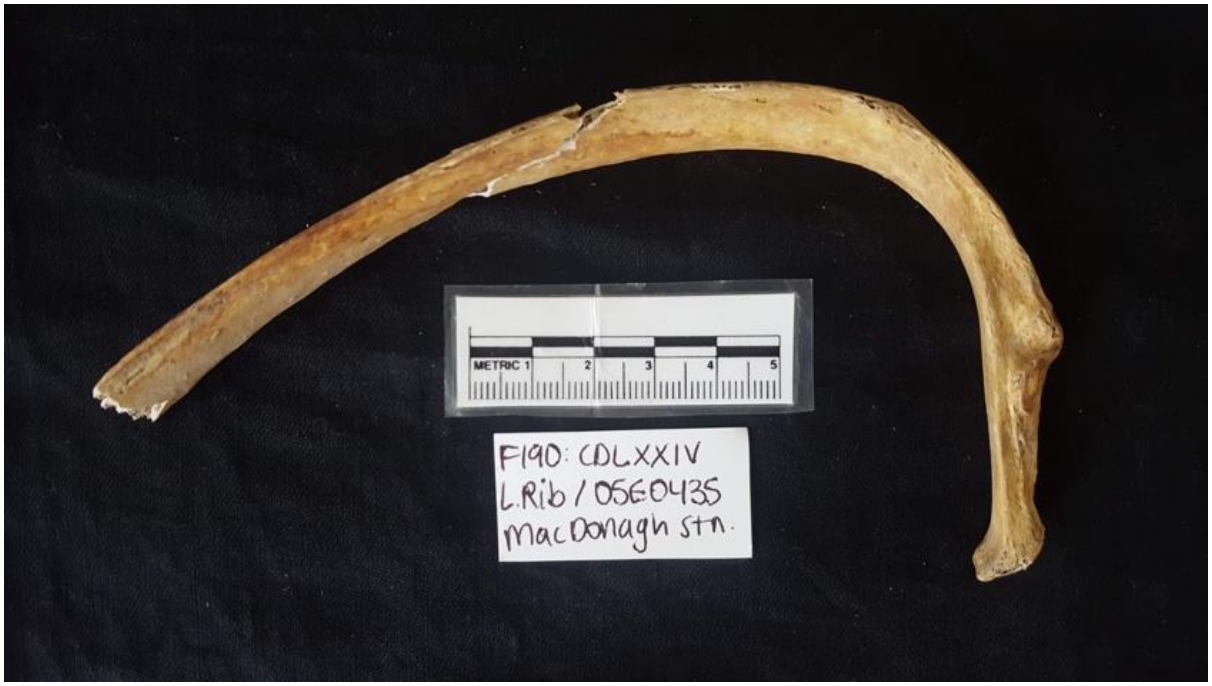


Figure 4.1. Middle rib of an adult female (CDLXXIV) prior to sampling

4.2. METHODS FOR HISTOLOGICAL ANALYSIS AND THEIR LIMITATIONS

The research questions explored in this thesis are primarily concerned with the response of bone microstructure to metabolic and infectious disease brought on by circumstances of structural violence leading up to, and throughout, the Great Famine. Therefore, it is in the best interest of this study to sample from a bone that is uninhibited by variations in dynamic mechanical stimuli.

The rib was chosen as the element for analysis for multiple reasons. First, ribs are often considered to be an ideal region for histological analysis of metabolic processes in bone because they are controlled by a static dynamic bending due to respiration which is less variable in its load pressure than limb bone diaphyses (Agnew and Stout 2012; Bellemare, Jeanneret, and Couture 2003; Robling and Stout 2003). As a result, when controlled for age (discussed in Section 2.5.2.), histomorphometric variation in rib bone microstructure from a known stressed population will more likely reflect metabolic changes due to health and diet rather than the effects of mechanical stimuli (Mulhern 2000). Additionally, the rib experiences high rates of bone turnover relative to other skeletal elements, potentially acting as an early detector of allostatic disruption (Fahy et al. 2017; Frost 1983). Studies have routinely showed a lack of

sexual dimorphism in the rib histology of individuals from "healthy" samples including percent cortical area, percent porosity area, and osteon area (Britz et al. 2009; Dominguez and Agnew 2016; Mulhern 2000), except in cases where elderly individuals are involved (Dominguez and Crowder, 2015). Therefore, differences observed between the sexes in samples with young individuals and known population history may be attributed to differential distribution of labor, access to resources, susceptibility to disease, or maternal processes like pregnancy or lactation (Dominguez and Agnew 2016; Goliath, Stewart, and Stout 2016; Stout and Lueck 1995).

However, there are limitations to histological analysis of cortical bone that impact not only investigations into the rib but other elements as well, including the state of bone preservation and the nature of destructive analysis. From a practical perspective, while ribs are likely to be represented in the archaeological record, they are often found in states of poor preservation including fracture and the presence of inclusions from diagenesis, known as histotaphonomy (Brickley 2006; Mays, Fysh, and Taylor 2002). This fact limits the utility of bone histological analysis since an entire cross section from the middle section of the rib is needed to collect many of the variables necessary for analysis, such as OPD which requires an entirely visible cross-section. While bone histology methodology is currently considered the "gold standard" for microscopic investigations into disease, diet, and nutrition (Holcombe et al. 2019), the destructive nature inherent in the preparation of bone sections for histological analysis also limits the elements available for study since archaeological skeletal material is scarce and often well protected (Holcombe et al. 2019). Cross-sections are viewed under the microscope from a two-dimensional perspective and only capture measured data from a single slice of the bone, which may not be representative of the overall bone quality or remodeling pattern. Unfortunately, widely available micro computed tomography (micro-CT) does not have the resolution quality capable of capturing the minute and specific histomorphometric variables necessary to answer research questions such as those presented in this study. However, the wide acceptance of data from cut sections strengthens the argument for destructive analysis (Holcombe et al. 2019). In this sample, the issue of preservation was mitigated by the large number of ribs available for destructive analysis, which was approved by the National Museum of Ireland.

The presence of regular respiratory mechanical loading, the relatively speedy rate of bone remodeling, and the practical features of the rib makes this skeletal element a prime candidate for histological analysis over other bony elements. This is fortunate, since the ribs were the only element approved for bone histological analysis of individuals from the Kilkenny Union Workhouse sample.

4.2.1. SAMPLE PREPARATION

After selecting the sample, the sectioned ribs were shipped to the Histology Laboratory in the Pathology Department at the Dunedin School of Medicine at the University of Otago in Dunedin, New Zealand where they were prepared for histological analysis according to the following procedure:

First, a release agent was applied to the internal surfaces of the EPDM 32mm mounting cups for easy removal of molds. Then, rib sections were placed in a transverse orientation so that the cut ends of the rib aligned with the walls of the cups. For sample identification, individual labels were placed in each cup with the label facing the external wall of the cup. The samples were then embedded in EpoThin^{TM} 2 Epoxy Hardener and Buehler® EpoThin^{TM} 2 Fast Cure Epoxy Resin following product instructions (Figure 4.2). Then, the sections were placed under a vacuum impregnation system to ensure all air was removed and resin infiltrated all the porous regions of the bone. This step improves sample stability throughout the sectioning and grinding process (Cho 2012). Once the embedding material has cured (about 12 hours) (Figure 4.3), two thin, 1-millimeter (mm) sections from each block were cut using a Buehler IsoMet® 1000 Precision Saw with a diamond blade (Figure 4.4).

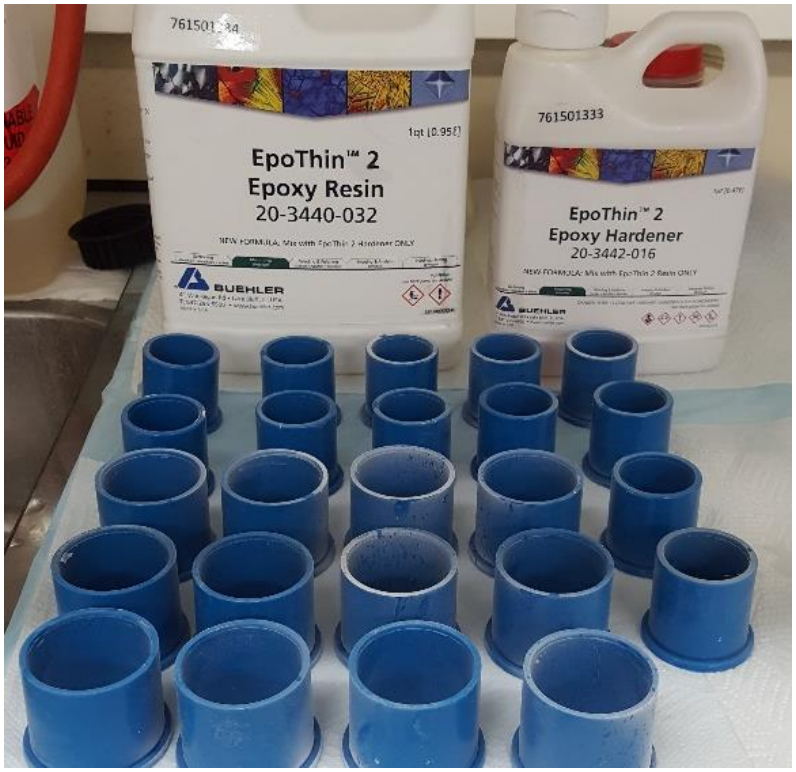


Figure 4.2. EpoThinTM 2 Epoxy Hardener and Buehler® EpoThinTM 2 Fast Cure Epoxy Resin with embedding cups

Figure 4.3. Rib samples embedded in resin blocks prior to sectioning

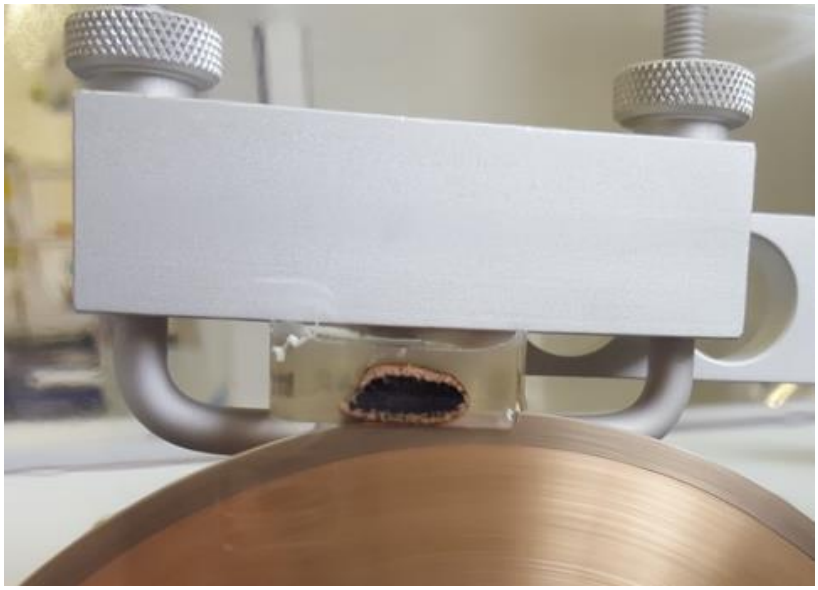


Figure 4.4. Rib sectioning on a Buehler IsoMet® 1000 Precision Saw with a diamond blade

Next, the thick sections were ground to between 50-100 um depending on when they were thin enough to clearly view the microstructure under bright and polarized light in the microscope. Grinding was done by hand on a MetaServ®3000 Variable Speed Grinder-Polisher by moving the sample in a back and forth and circular motion between an abrasive paper (240 grit) and a rotating UltraPrep Metal Bonded Diamond Disc. After grinding, the samples were placed in a bath of Histo-Clear (a xylene substitute that assists in the removal of superficial diagenesis) for five to ten minutes depending on the degree of taphonomic alteration.

Finally, sections were polished using a WhiteFelt Premium Polishing Cloth and MetaDi Fluid and MasterPrep Polishing Suspension to protect the sample and remove diamond and abrasive paper residue. Once the sample was embedded, cut, ground, placed in Histo-clear, and polished to an appropriate thickness, the bone was placed in a folded piece of tissue between two microscope slides. This process allowed the samples to dry and remain flat overnight in preparation for mounting. The following day, the thin rib sections were carefully secured to a glass microscope with DPX mounting medium and a cover slip. Then, slides were placed on a flat slide tray to dry for at least one day before analysis. The slides are stored this way until they are completely dry to prevent the section from sliding under the glass and lifting the cover slip. Once dry, each rib section was imaged using an Aperio C32 Leica slide scanner at 20X

magnification in bright light and a Leica DM6M LED scanning microscope at 20X magnification in polarized light.

4.2.2. ANALYSIS OF THE PRESERVATION OF THE RIBS

After preparation, all samples were observed under the microscope and scored according to the Oxford Histological Index (OHI) (Table 4.2) and Birefringence Index (BI) (Table 4.3) to identify the quality of preservation. Due to the rib's location in the body and its thin cortex, these bones are inherently susceptible to diagenetic changes, the taphonomic alterations that can inhibit the visibility of the microstructure after death. For example, unless a recently deceased body has been disarticulated or scavenged, it will almost always go through putrefaction, one of the earliest stages of decomposition. During this stage, the body enters autolysis, causing soft tissue organs to liquefy and gases to build up within the thorax until they are finally released through the eyes, mouth, ears, nose, and other orifices (Gleiber et al. 2017). The ribs are on the front lines throughout this experience and are likely to annex micro-organisms like bacteria as they soak through the periosteum and into the medullary cavity of highly porous or fractured bones (Booth, Redfern, and Gowland 2016; Hedges, Millard, and Pike 1995; Jans 2008). Even if the rib survives the initial threat of abdominal microbial intrusion, it has the potential to become infiltrated by fungi or bacteria due to waterlogging in the anoxic environment of a poorly irrigated grave. Unfortunately, taphonomically unaltered microstructure is rare in historic and archaeological human and non-human bone samples (Jans 2008).

The extent of taphonomic change in each rib section was documented to determine which slides were suitable to collect histomorphometric variables. This was accomplished using the Oxford Histological Index (OHI) and the Birefringence Index (BI) (Figures 4.5 and 4.6). OHI is an ordinal measure of the amount of bone that is visible or obstructed by taphonomic change (i.e., biogenesis, chemical diagenesis) and is recorded on an ordinal scale from 0-5; while BI is an ordinal measure of the amount of birefringence in the cross-section under polarized light, usually visible through the contrast of the collagen and mineral components in bone. BI is scored as 0, 0.5, or 1 depending on the visibility of osteons and their structures within the cortex (Booth, Redfern, and Gowland 2016; Hackett 1981; Hedges, Millard, and Pike 1995; Jans et al. 2004).

When collagen leaches from the bone because of bioerosion or waterlogging, clarity of structures within the cortex of the cross section decreases and the BI score is lowered. Qualitative observations of staining and inclusion within the microstructure were also recorded. This information will be useful for future investigations of the rib from this sample and comparative analysis of the effect of waterlogging on the histological preservation of historic skeletal material discovered in mass graves. The amount of visibility of histological structures in the cross section is important because some variables, such as OPD and DZ (described below), require an entire cross section of bone.

It is important to note that because preservation of bone is correlated to natural bone porosity, there is likely to be bias in the sampling procedure wherein sampling occurs from a region of the burial ground with younger or "healthier" individuals (Jans et al. 2004).

Table 4.2. Oxford Histological Index values (Hedges et al. 1995; Millard 2001; Booth 2017)

Index	% of Intact Bone	Description
0	<5%	No original features identifiable, other than Haversian canals
1	<15%	Small areas of well-preserved bone present, or some lamellar structure preserved by pattern of destructive foci
2	<50%	Some well-preserved bone present between destroyed areas
3	>50%	Larger areas of well-preserved bone present
4	>85%	Bone is fairly well preserved with minor amounts of destroyed areas
5	>95%	Very well preserved, similar to modern bone

Table 4.3. Birefringence Index values (Jans et al. 2004; Booth 2017)

Index	Birefringence	Description
0	Obliterated	No birefringence can be seen under polarized light
0.5	Reduced	Birefringence can be seen in some of the microstructure under polarized light
1	Normal	All or most osteons are bright and visible under polarized light

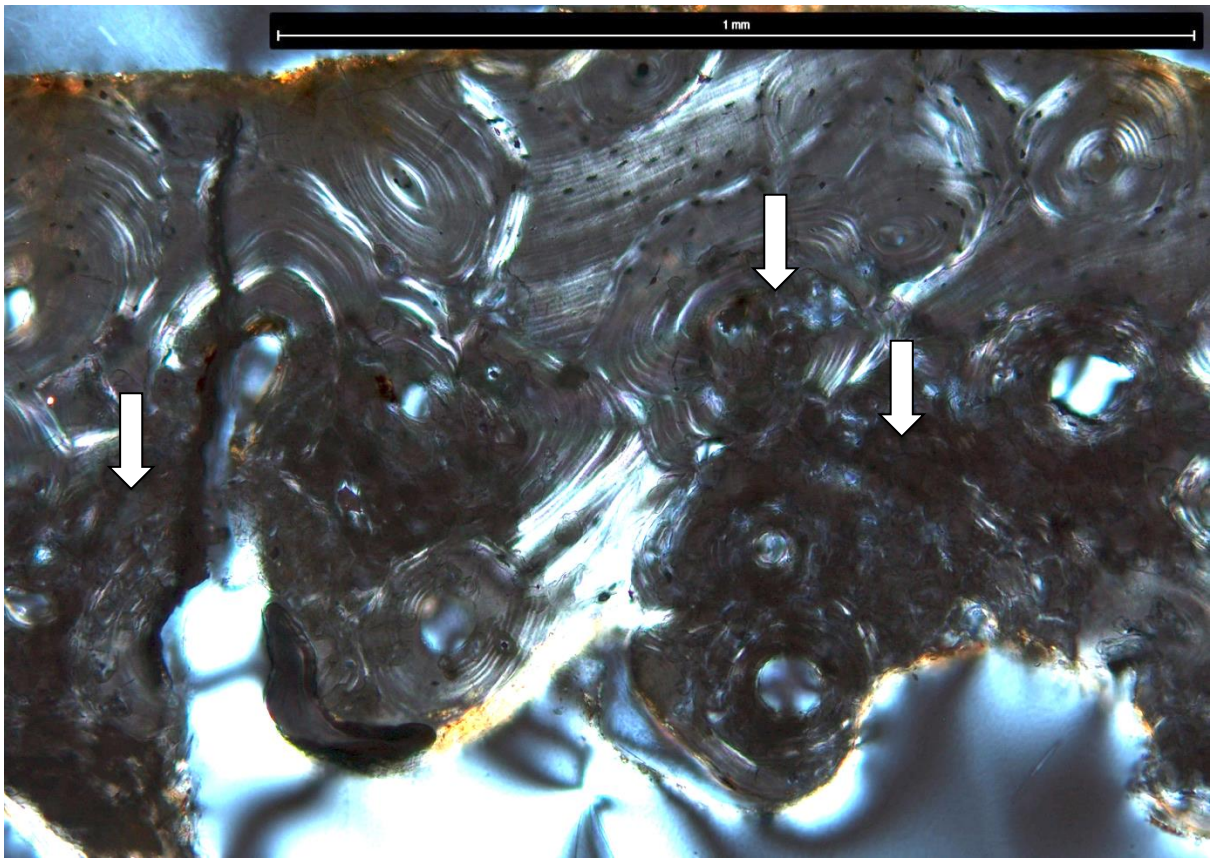


Figure 4.5. Diagenesis (white arrow) in the middle rib of an early middle adult female (CCXCVII) with a BHI score of 3 indicating large areas of well-preserved bone and BI score of 1 indicating most osteons are bright under polarized light (10x magnification under polarized light; scale set at 1 mm)

Figure 4.6. Diagenesis throughout the middle rib of a young child with a point age estimate of 1.3 years old with an OHI of 0 indicating complete obliteration of bone structures and a BI score of 0 indicating no birefringence when viewed under polarized light (10x magnification under polarized light; scale at 1 mm)

4.2.3. RIB BONE HISTOLOGICAL VARIABLES TO BE MEASURED

After the slides are scanned, photographed, and the ribs were scored for preservation, the histomorphometric data for all variables used to interpret bone health (Table 4.4) were collected manually using the ImageJ 1.52a software (Rasband 2013) and a Wacom Intuos Pro drawing tablet and a digitizing pen. The histomorphometric variables measured for the adult include: Total area (Tt.Ar), Endosteal area (Es.Ar), Cortical area (Ct.Ar), Percent cortical area (%Ct.Ar), Porosity area (Po.Ar), Percent porosity area (%Po.Ar), Osteon area (On.Ar), Osteon circularity (On.Cr), Haversian canal area (Ha.Ar), Haversian canal circularity (Ha.Cr), Osteon Population Density (OPD), and Double zonal osteons (DZ). For the subadult sample, all the same variables were measured except for double zonal osteons. Primary canal area and circularity (Pr.Ar; Pr.Cr) measurements were included for subadults. This following section describes the value of examining these variables.

Table 4.4. Definitions of histomorphometric variables. Adapted from Parfitt 1994 (*used in analysis)

Histomorphometric Variable	Abbreviation	Description
Total area	Tt.Ar	Total cross-sectional area within the periosteal border
Endosteal area	En.Ar	Area within the endosteal border or the marrow cavity
Cortical area	Ct.Ar	Area of cortical bone between the periosteal and endosteal borders (Tt.Ar - En.Ar)
*Percent cortical area	%Ct.Ar	Percent cortical area relative to the total area (Ct.Ar*100/Tt.Ar)
*Porosity area	Po.Ar	Area of all porosity within the cortical bone
*Percent porosity area	%Po.Ar	Intracortical porosity relative to total cortical area (Po.Ar*100/Ct.Ar)
*Osteon area	On.Ar	Total area within the reversal line of an intact osteon
*Osteon circularity	On.Cr	Shape of the osteon relative to a true circle
*Haversian canal area	Ha.Ar	Total area within the Haversian canal of an intact osteon
*Haversian canal circularity	Ha.Cr	Shape of the Haversian canal relative to a true circle
Intact osteons	N.On	Number of secondary osteons with an intact Haversian canal bound by a scalloped reversal line
Fragmented osteons	N.Fg.On	Number of fragmented osteons having a visible Haversian canal that has been partially resorbed
*Osteon population density	OPD	Sum of secondary intact and fragmented osteons within the cortex
*Double zonal osteons	DZ	Bright ring of hyper-mineralization in an osteon under polarized light

4.2.3.1. Cortical area and cortical porosity area

Cortical area and cortical porosity area are essential variables collected in the histological analysis of cortical bone from the Kilkenny Union Workhouse skeletal sample because they are representations of bone strength or fragility (Paine and Brenton 2007; Cho and Stout 2011). As discussed in Chapter 2, for bone to maintain structural integrity the material processes of bone must adhere to a regular pattern of bone turnover. For example, osteoclastic resorption initiated by osteocyte signaling must be followed by osteoblast deposition that is then mineralized into new bone. However, this homeostatic process may be altered by metabolic disruptions, such as the accelerated initiation of osteocyte resorption by the parathyroid hormone in hyperparathyroidism (McCarthy 2016) or the reduction of osteoid deposition in individuals with Vitamin C deficiency (Snoddy et al. 2018), both issues that may cause extensive cortical pores that are not filled in resulting in fragile cortices.

Total area (Tt.Ar) is the total cross-sectional area within the periosteal border. This variable is necessary for quantifying cortical bone area within the cross section. Since total area encompasses the cortex and the medullary cavity, calculation of cortical area (Ct.Ar) from total area is necessary to reveal how much of the cross section is within the periosteum but outside the endosteum. To determine the cortical area, endosteal area (Es.Ar) is measured and subtracted from total area (Figure 4.6). Porosity area describes how much of that bony cortex is composed of voids or absent of bone due to remodeling events taking place at the time of death (Figure 4.7). Porosity area (Po.Ar) is calculated by figuring the sum of the cortical pores. While porosity is a natural feature of cortical remodeling, the coupled action of osteoclasts and osteoblasts in metabolically healthy bone ensures that porosity exists in a homeostatic balance to maintain hardness and stiffness properties (Stout, Cole, and Agnew 2019). When this balance is disrupted by inadequate nutrition, reproductive stress, or hormonal changes, increases in porosity can occur that lead to increased risk of fracture (Stout, Cole, and Agnew 2019).

Cortical area and porosity area in the rib are variables that have been shown to follow an age-related pattern regulated by biomechanical loading and genetic factors. For example, healthy subadults are known to have large and many pores, whereas porous bones in adults may indicate osteoporosis or an imbalance in bone remodeling. Due to this difference, adults and subadults will be analyzed separately so that differences in disease processes may be more clearly observed (Pfeiffer et al. 2006; Streeter 2010). For this study, cortical and porosity area were used to calculate the percent of cortical area (%Ct.Ar) and percent cortical porosity area (%Po.Ar) to control for allometry between individuals while informing how much of the cross section is made up of cortical bone, and what percent is porotic (Dominguez and Agnew 2016; Martin, Magennis, and Rose 1987). If the entire cortex was not present, these variables were not calculated.

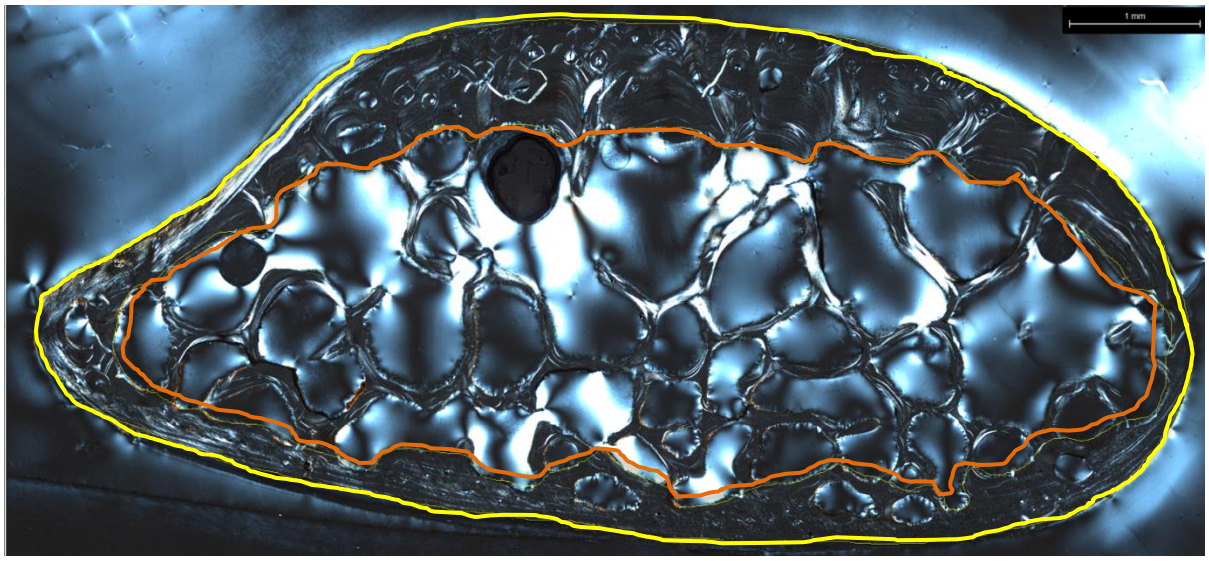


Figure 4.7. Cortical area of the rib calculated by subtracting endosteal area (inner orange outline) from total area (outer yellow outline) (10x magnification under polarized light; scale set at 1 mm)

4.2.3.2. Osteons, Haversian canals, OPD, and double zonal osteons

Osteon population density (OPD) is based on the accumulation of secondary osteons in the cortex due to continued remodeling activity. Osteons begin accumulating in the rib beginning in childhood around the age of 12.5, known as the effective age of adult compacta (Stout and Lueck 1995; Wu et al. 1970), and continue to grow in number as remodeling occurs throughout adulthood (Streeter 2010; Stout and Paine 1992). However, these mature features of bone are not static in their morphology throughout life and many researchers have observed that both osteons and Haversian canals become smaller and more circular over time which may have biomechanical, metabolic, or geometric implications (Goliath, Stewart, and Stout 2016; Landeros and Frost, 1960; Paine and Godfrey 1996; Takahashi, Epker, and Frost 1965). Figure 4.7 shows a resorption bay measured for porosity area and percent porosity area, and osteons and a Haversian canal measured for area and circularity.

In cases where new osteons (and smaller) are not being created, OPD will also be impacted. Since osteon size (On.Ar) and shape (On.Cr) are correlated to Haversian canal size (Ha.Ar) and shape (Ha.Cr) and OPD, each of these variables are used in this thesis to investigate the effect of metabolic disruption on rib bone histology of the Kilkenny Union Workhouse population sample. For example, those who died while suffering from osteomalacia or childhood rickets may have hypo-mineralized regions of bone where osteoid was deposited but not mineralized, prohibiting secondary osteon formation and remodeling in adolescents and adults. These

individuals are likely to have fewer, larger, and less circular osteons and Haversian canals if resorption is occurring but new osteons and associated Haversian canals are not forming.

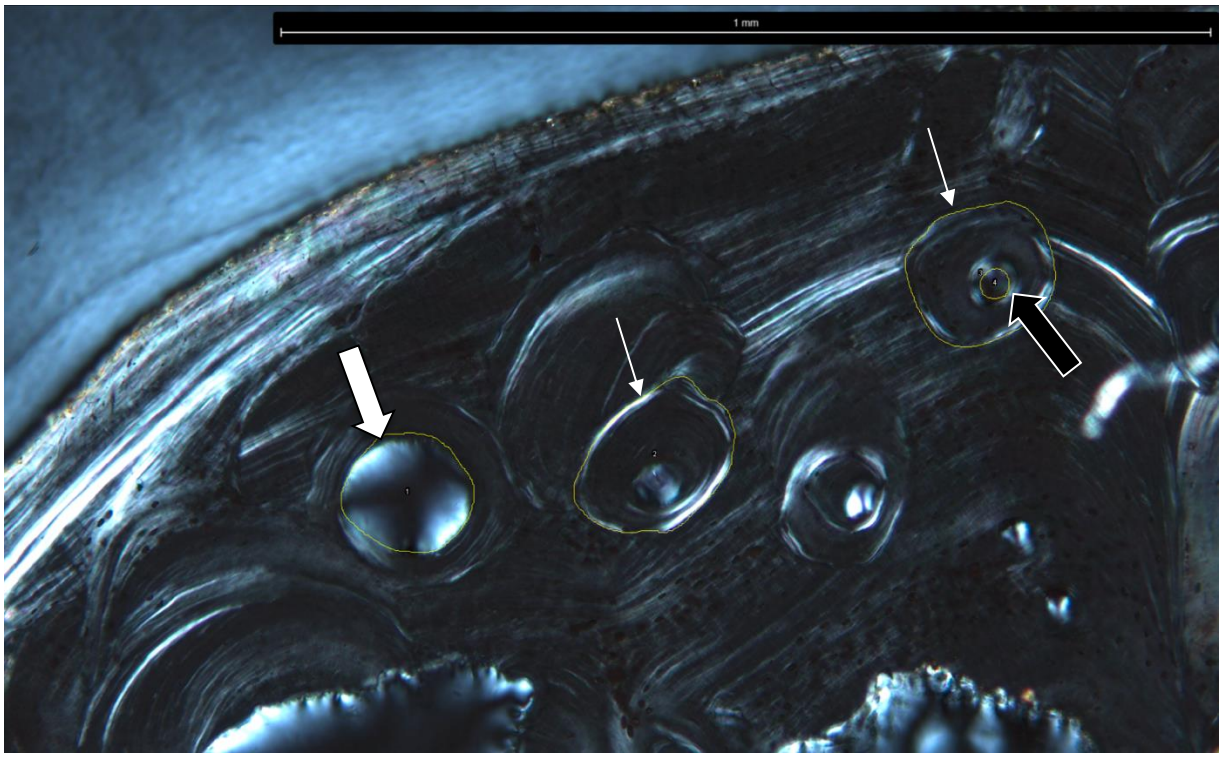


Figure 4.8. Variables measured for histomorphometry (outlined in yellow): Porosity (large white arrow); intact osteons (small white arrow) C) Haversian canal (black arrow) (10x magnification under polarized light; scale set at 1 mm)

Additionally, double zonal osteons, observed as osteons with at least one hyper-mineralized ring of concentric lamellae, were counted in adult ribs with complete cross-sections (Raguin and Streeter 2018). These osteons are thought to be artefacts of arrested mineral deposition and may indicate recent episodes of recovery from metabolic stress (Raguin and Streeter 2018). If mineralization was halted, as it is in individuals suffering from osteomalacia, there may be more double zonal osteons in these individuals.

Ideally, at least fifty osteons and Haversian canals are necessary to obtain average area and circularity. To obtain porosity, OPD, and double zonal counts, the entire cross section is necessary. However, sometimes the whole cross section was not visible due to diagenesis or fractures. In this case, the latter two variables could not be properly measured.

4.2.4. STATISTICAL ANALYSES

Each of the variables used in this thesis and discussed in the previous section will be helpful for informing whether homeostatic remodeling was altered in rib bones of the people who were buried on the Kilkenny Union Workhouse grounds when metabolic disruption occurred due to malnutrition and/or disease, and when relief food (maize) was provided as a dietary supplement.

The distribution of metabolic disease (Table 4.5) and infectious disease (Table 4.6) separated by age cohort and presented in the sample are based on the paleopathological analysis by Geber (2015) described in Chapter 3. The results of those paleopathological analyses have been peer-reviewed by academic journals and book publishers of high standing, indicating the diagnoses, where possible, are valid and the individuals without lesions (Geber and Murphy 2012; Geber 2014; Geber 2015; Geber 2016). Of those that were included in this sample and show evidence of metabolic disease (72%), adults and subadults combined have lesions indicative of scurvy (58.6%), rickets/osteomalacia (4.8%), and the rest have non-specific lesions indicative of metabolic stress, such as osteopenia (1.6%) and rib lesions (7.5%). Of those that show evidence of infectious disease (28%), adults and subadults combined had sinusitis (7%), followed by tuberculosis (2.2%), syphilis (1.1%), osteomyelitis (1.1%), and the remainder had non-specific lesions that could be indicative of infectious disease, such as rib lesions which may indicate respiratory disease but could also be indicative of metabolic diseases like scurvy, mentioned above.

Diseases were grouped by type (i.e., “No Lesions”, “Only Infectious”, “Only Metabolic”, or “Metabolic and Infectious”) due to the small sample sizes for each disease diagnosis (Tables 4.7-4.10). For example, all individuals with lesions indicative of infectious disease (sinusitis, tuberculosis, syphilis, osteomyelitis, etc.) were collated together in the “Only Infectious” disease group. If there were lesions indicative of both metabolic and infectious disease, the individual would be classified in the “Metabolic and Infectious” disease group.

Table 4.5. Crude prevalence rate of metabolic diseases identified within the Kilkenny Union Workhouse population sample (n) and population (N)

	Metabolic Disease							
	Osteopenia		Rickets/Osteomalacia		Scurvy		Rib Lesions	
	n (%)	N (%)	n (%)	N (%)	n (%)	N (%)	n (%)	N (%)
Adults	3	6.6	6.1	5.8	43.4	39.8	7.1	7.5
Subadults	0	0	3.4	4	75.9	60.6	8	5.1
Combined Age Groups	1.6	2.9	4.8	4.8	58.6	51.4	7.5	6.2

Table 4.6. Crude prevalence rate of infectious diseases identified within the Kilkenny Union Workhouse population sample (n) and population (N)

	Infectious Disease							
	Sinusitis		Tuberculosis		Osteomyelitis		Syphilis	
	n (%)	N (%)	n (%)	N (%)	n (%)	N (%)	n (%)	N (%)
Adults	12.1	8	3	.9	1	1.4	2	.5
Subadults	1.1	1.7	1.1	.6	1.1	.6	0	.2
Combined Age Groups	7.5	4.4	2.2	.7	1.1	.9	1.1	.3

Table 4.7. Descriptive statistics for adults in disease groups in the overall population (N) of skeletal remains from the Kilkenny Union Workhouse population sample (age is in years)

	N	*N* (%)	Female	Mean Age	Male	Mean Age	Ind.	Mean Age
Only Infectious	19	4.5	10	36.3	9	39.6	0	-
Only Metabolic	170	40.2	70	37.6	96	40.4	4	-
Infectious and Metabolic	48	11.3	17	36.9	30	38.4	1	-
None	187	44	99	35.6	77	39	11	-
Total	424	100	196	36.6	212	39.4	16	-

Table 4.8. Descriptive statistics for adults in disease groups of skeletal remains used in this thesis (n) from the Kilkenny Union Workhouse population sample (age is in years)

	n	*n* (%)	Female	Mean Age	Male	Mean Age
Only Infectious	9	9.1	7	38	2	41.4
Only Metabolic	43	43.4	19	36.4	24	37.9
Infectious and Metabolic	12	12.1	6	34.8	6	38.9
None	35	35.4	23	34.5	12	38.5
Total	99	100	55	35.9	44	39.2

Table 4.9. Descriptive statistics for subadults in disease groups in the overall population (N) of skeletal remains from the Kilkenny Union Workhouse population sample (age is in years)

	N	*N* (%)	Mean Age
Only Infectious	11	2	6.2
Only Metabolic	335	61.6	5.2
Infectious and Metabolic	30	5.5	9.2
None	168	30.9	4.3
Total	544	100	6.2

Table 4.10. Descriptive statistics for subadults in disease groups of skeletal remains used in this thesis (n) from the Kilkenny Union Workhouse population sample (age is in years)

	n	*n* (%)	Mean Age
Only Infectious	3	3.4	14
Only Metabolic	62	71.3	8
Infectious and Metabolic	7	8.1	12.1
None	15	17.2	10
Total	87	100	13.3

4.2.4.1. Descriptive statistics and age correlations

All statistical analyses were performed using SPSS 26.0 (IBM Corp., 2013). The data was tested for normality using a Shapiro-Wilk test and normalized using natural logarithm if necessary. Once all data were normalized, a *t*-test was used to test for differences between the sexes for each variable. Then, since percent cortical area, percent cortical porosity, OPD, and osteon and Haversian canal size and shape have all been shown to remodel in a predictable pattern over time (Dominguez and Agnew 2016; Goliath, Stewart, and Stout 2016; Stout and Paine 1992), a Pearson Correlation Coefficient was performed to determine if there is a relationship between age and bone histological variables for the Kilkenny Union Workhouse population sample. A Shapiro-Wilk test for normality was used and when the data was not normal a natural logarithm was performed before further analyses are conducted. A Levene's Test was used to check for equal variances to be sure all data are homogenous. Intra-observer error was tested using a T-test for differences between histomorphometric data from five individuals collected at least six months after the original data set was analyzed.

4.2.4.2. Statistical analyses for bone histology

Since males and females may have had different resource requirements depending on their age, labor, and parity status, an independent t-test was used to test for sex differences for each variable in the adult cohort. To address the hypotheses listed under Question 1 (Section 1.2.),

an initial t-test was employed to determine if there are differences in histomorphometry between each disease type for both the adult and subadult cohorts. Then, an analysis of covariance (ANCOVA) was conducted to determine if the observed differences in histomorphometry between disease types changed when age is included as a covariate.

Geber found evidence of scurvy in 169 adult and 330 subadult skeletons from the Kilkenny Union Workhouse burials. In the rib subsample used for this thesis, 43 derive from adults (43.4%; 43/99) and 66 from subadults (75.9%; 66/87) diagnosed with scurvy. Since evidence of scurvy was prevalent in this sample, individuals with scurvy were compared to those without scurvy to determine if the presence of scurvy has an impact on bone histomorphometry. To control for histological changes due to comorbidities with infectious disease, adults with only metabolic lesions were compared, then those within the metabolic and infectious disease were compared, followed by all groups combined (Table 4.11). Of all adults with only metabolic disease, 74.4% had lesions indicative of scurvy (32/43) while 25.6% did not have scorbutic lesions (11/43). In the metabolic and infectious disease group 83.3% had lesions (10/12) while 16.6% did not (2/12). When all diseases are combined, 76.4% had lesions indicative of scurvy (42/55) while 23.6% did not (13/55).

In the subadult cohort those with only metabolic disease had 93.5% individuals with lesions indicative of scurvy (58/62) and 6.5% did not have scurvy lesions (4/62). There were no individuals without evidence of scurvy in the metabolic and infectious disease group in this cohort, so this group was not analyzed. When all disease types are combined, 92.7% had lesions (64/69) and 5.8% did not have scurvy lesions (4/69) (Table 4.7).

Table 4.11. Descriptive statistics of adults with and without lesions indicative of scurvy

Disease group	Scurvy		Total
	Not Present	Present	
Only metabolic disease	11	32	43
Metabolic and infectious disease	2	10	12
All disease types	13	42	55

Table 4.12. Descriptive statistics of subadults with and without lesions indicative of scurvy

Disease group	Scurvy		Total
	Not Present	Present	
Only metabolic disease	4	58	62
Metabolic and infectious disease	0	7	7
All disease types	4	64	69

All nine adults used for light stable isotope analysis have lesions indicative of metabolic disease and two showed comorbidities of metabolic and infectious disease. The isotope analyses conducted by Beaumont and colleagues identified three children with $\delta^{13}C$ in the C_3 range while six children showed C_4 values in their bulk rib bone collagen. The latter data supports historical literature stating that maize was provided as a relief food in the Kilkenny Union Workhouse (Beaumont et al. 2013). To explore Question 2 (Section 1.2.), a Pearson Correlation Coefficient test was conducted to look at the strength of the relationship between the histomorphometric data and $\delta^{15}N$ and $\delta^{13}C$ isotopes from the ribs of individuals in both the adult (Tables 4.8) and subadult (Table 4.9) cohorts. Mean stable isotope values were obtained from analyses conducted by Julia Beaumont (Beaumont et al. 2013).

Table 4.13. Mean stable isotope values ($\delta^{15}N$ ‰ and $\delta^{13}C$ ‰) for subadults from the Kilkenny Union Workhouse population sample

	N	Minimum	Maximum	Mean	Std. Deviation	Min. Age	Max. Age
$\delta^{13}C$ ‰	9	-20.3	-15.1	-17.711	1.6818	4	4
$\delta^{15}N$ ‰	9	8.7	10.6	9.800	.5916	9	9

Table 4.14. Mean stable isotope values ($\delta^{15}N$ ‰ and $\delta^{13}C$ ‰) for adults from the Kilkenny Union Workhouse population sample

	N	Minimum	Maximum	Mean	Std. Deviation	Min. Age	Max. Age
$\delta^{13}C$ ‰	17	-20.8	-17.2	-19.541	.9766	20	20
$\delta^{15}N$ ‰	17	9.5	11.6	10.847	.5580	51	51

4.3 CHAPTER SUMMARY

This chapter described the materials and methods that were used for the histological analysis of ribs obtained from adults and subadults from the Kilkenny Union Workhouse mass burials. The distributions of age and sex in the sample obtained for this thesis are representative of the larger sample excavated from the site in Kilkenny City. Once the ribs were scored for quality of

preservation the final sample size was 186 individuals (99 adults and 87 subadults). The next chapter is a report of the statistical analyses described above.

CHAPTER 5. RESULTS

This chapter describes the results obtained using the methodology described in Chapter 4. The first section includes the results for preservation scoring (OHI and BI) that were used to determine the final sample size, then the results for normality and equal variances testing for the histomorphometric variables are shown followed by sex differences observed within the sample and correlations with age for each cohort. Finally, the results for the two research questions are presented.

5.1. PRESERVATION OF THE RIBS AND DESCRIPTIVE STATISTICS

When preservation was evaluated, adult mean OHI was lower than subadult mean OHI but subadults had higher birefringence than adults (Table 5.1). After internal bone preservation was observed, those with a mostly visible microstructure (OHI > 3; BI > 0.5) were used for analysis and the subsample size was reduced to a grand total of 186 individual ribs (as described in Section 4.1). The OHI and BI scores for all individuals are listed in Appendix 1.1 and 1.2.

Table 5.1. Mean preservation scores for OHI and BI in the subsample from the Kilkenny Union Workhouse population sample

	n	OHI	BI
Adults	172	3.16	.71
Subadults	139	3.46	.65
Total	311	3.31	.68

5.2. SHAPIRO WILK TEST FOR NORMALITY, TEST FOR EQUAL VARIANCES, AND INTRA-OBSERVER ERROR

A Shapiro-Wilk test for normality indicated both On.Ar ($W(5) = .766$, $p = .04$) and Ha.Ar ($W(5) = .665$, $p = .004$) were not normally distributed. Data for On.Ar and Ha.Ar were normalized using a natural logarithm before further analysis. Intra-observer error reports showed no statistically significant differences between variables for five slides measured six months apart.

5.3. SEX DIFFERENCES IN HISTOMORPHOMETRY

Overall adult females exhibited significantly greater percent cortical area than adult males ($t(58)=3.32$, $p=.002$) (Table 5.2). There were no statistically significant differences for any other variables. Due to these results, sexes were pooled for all future analyses except %Ct.Ar, where sexes were first analyzed together in order to examine the overall effect of disease on %Ct.Ar, and then separately to better understand the specific experiences of females and males in the workhouse. Sex could not be estimated for all but two individuals in the subadult cohort, so subadults were not tested for sex differences.

Table 5.2. Comparison of %Ct.Ar between adult females and adult males (p=.002)

	Sex	N	Mean (mm^2)	SD
%Ct.Ar	Female	36	43.16	10.68
	Male	24	34.60	8.19

5.4. CORRELATIONS BETWEEN HISTOMORPHOMETRIC VARIABLES AND AGE

A Pearson Correlation Coefficient was used to test each variable for correlations with age to better understand the effects of disease on the rib histomorphometry of those who were buried on the grounds of the Kilkenny Union Workhouse. The results are presented in this section by cohort.

5.4.1. CORRELATIONS WITH AGE AND ADULT HISTOLOGY VARIABLES

A Pearson Correlation Coefficient showed a significant negative relationship between age and %Ct.Ar for the sexes combined ($r(58) =-.441$, $p< .001$) (Figure 5.1), females ($r(34) =-.394$, $p =.02$) (Figure 5.2), and males ($r(22) =.568$, $p =.004$) (Figure 5.3). There is also a significant negative relationship between age and On.Ar ($r(97) =-.282$, $p =.005$) (Figure 5.4), as well as a significant positive relationship between age and On.Cr ($r(97) =.250$, $p =.01$) (Figure 5.5), Ha.Cr ($r(97) =.251$, $p =.01$) (Figure 5.6), and OPD ($r(60) =.285$, $p =.03$) (Figure 5.7). While there is a significant relationship between age and five of the variables in the adult sample, the r value of each variable is between .5 and -.5. In a Pearson Correlation, the closer the r value is to 1 or -1 the stronger the association is between the two variables. The low r value indicates that although there is limited association for these variables, the variance may not be explained by age alone. Neither variable for porosity (Po.Ar; %Po.Ar) showed a linear correlation with age as expected based on previous research (Dominguez and Agnew 2016).

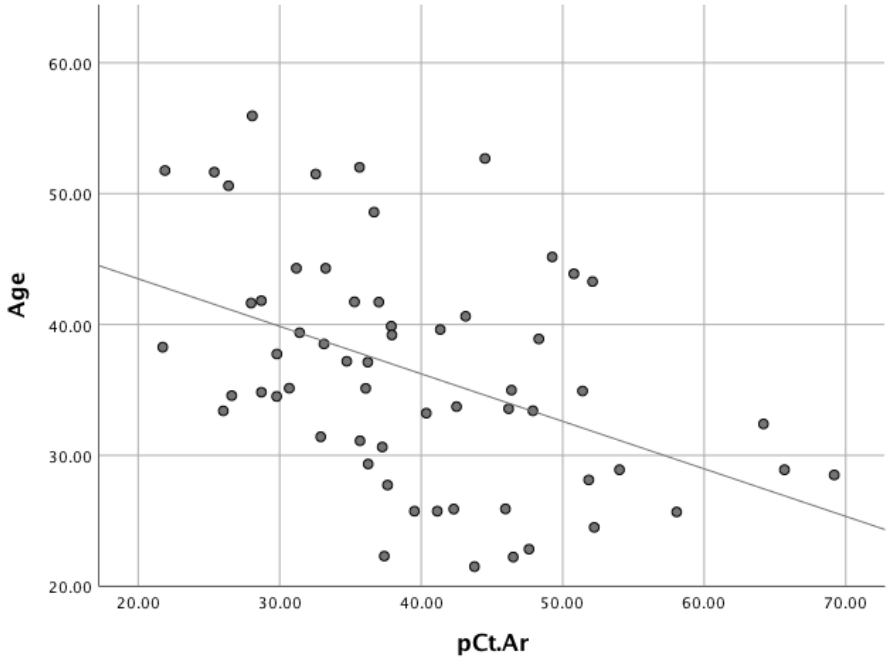


Figure 5.1. Scatterplot showing the linear relationship between age (years) and combined %Ct.Ar

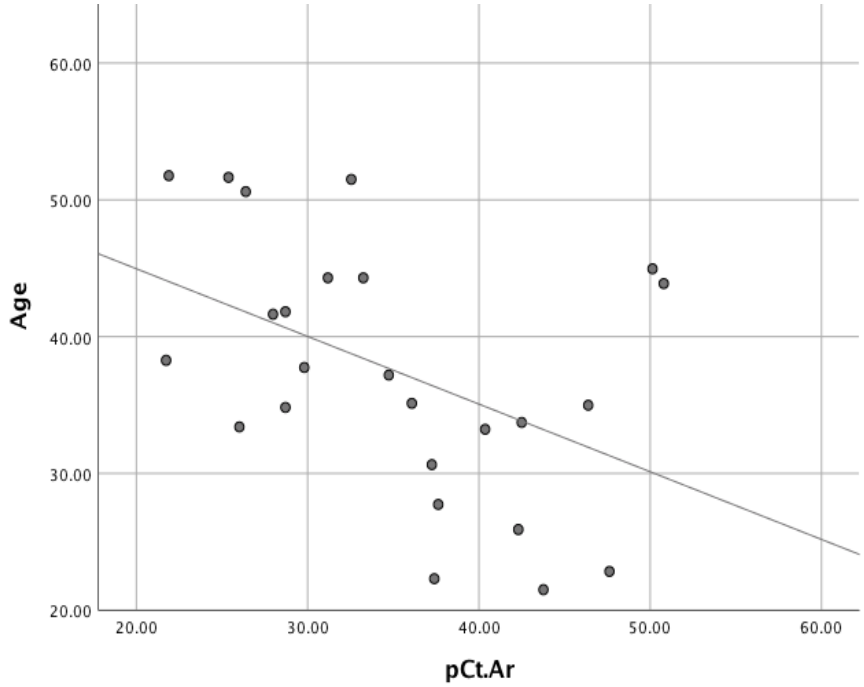


Figure 5.2. Scatterplot showing the relationship between age (years) and %Ct.Ar in adult females

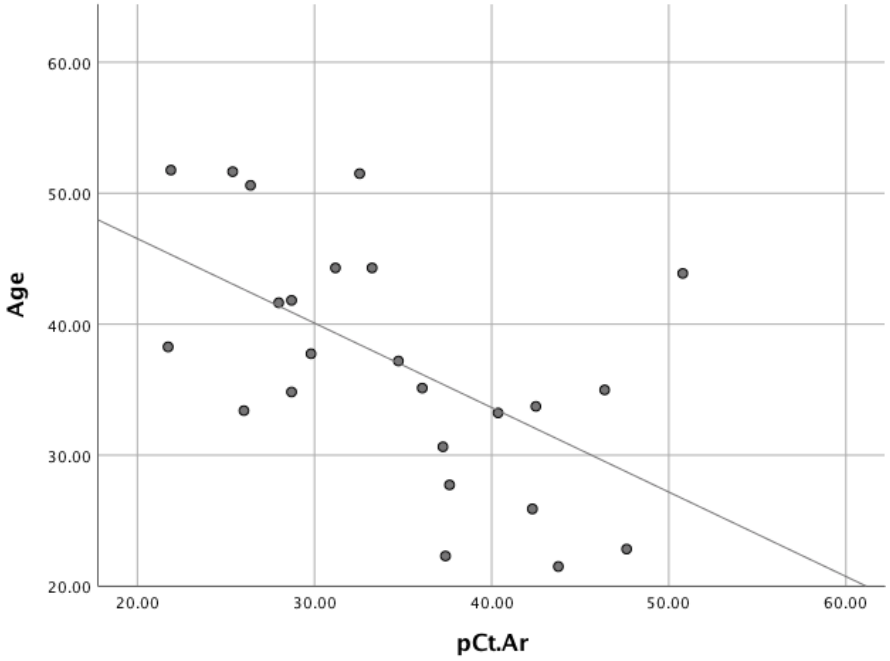


Figure 5.3. Scatterplot showing the relationship between age (years) and %Ct.Ar in adult males

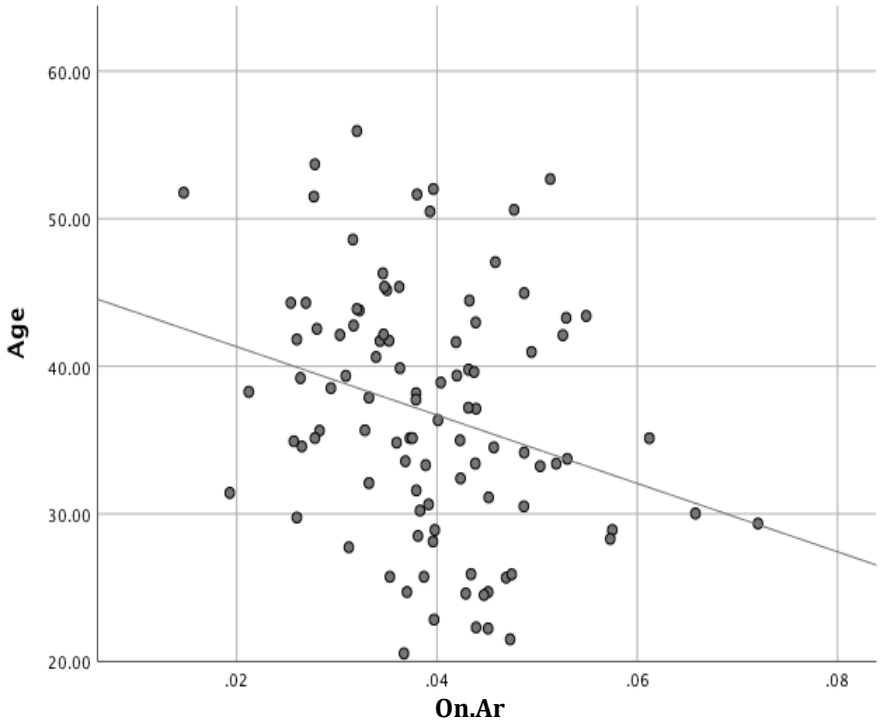


Figure 5.4. Scatterplot showing the relationship between adult age (years) and On.Ar

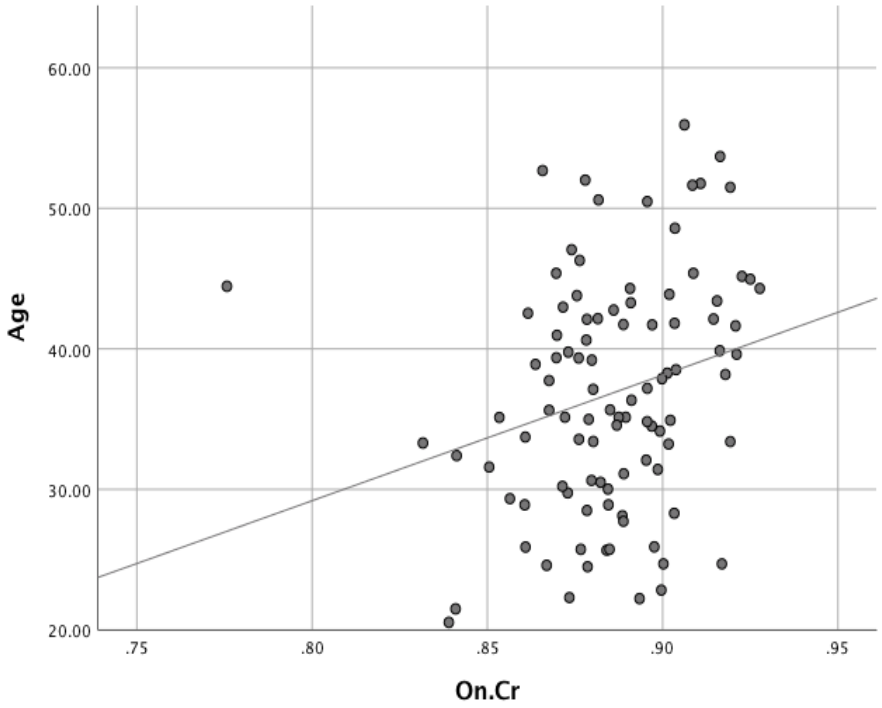


Figure 5.5. Scatterplot showing the relationship between adult age (years) and On.Cr

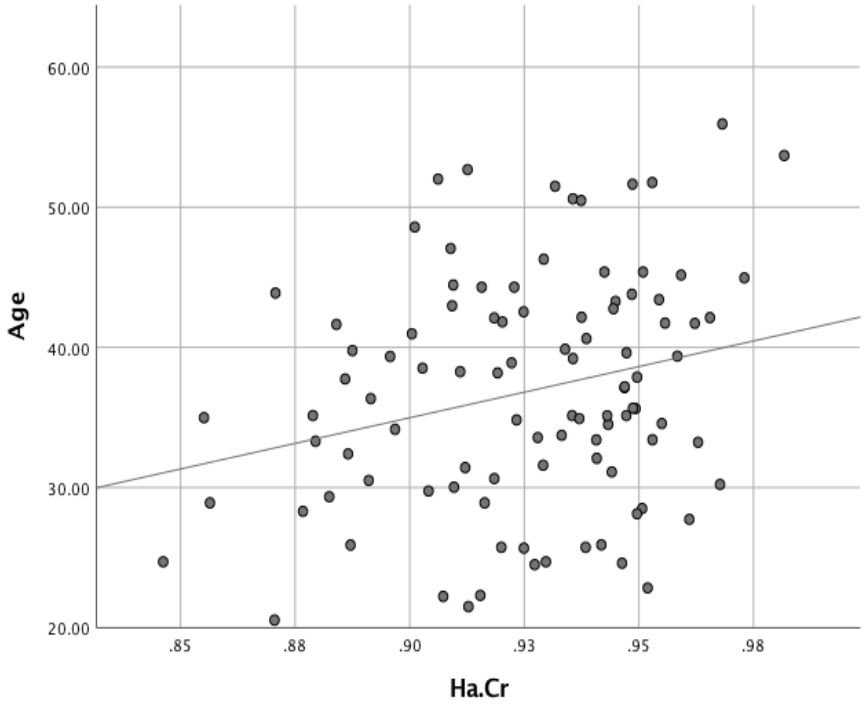


Figure 5.6. Scatterplot showing the relationship between adult age (years) and Ha.Cr

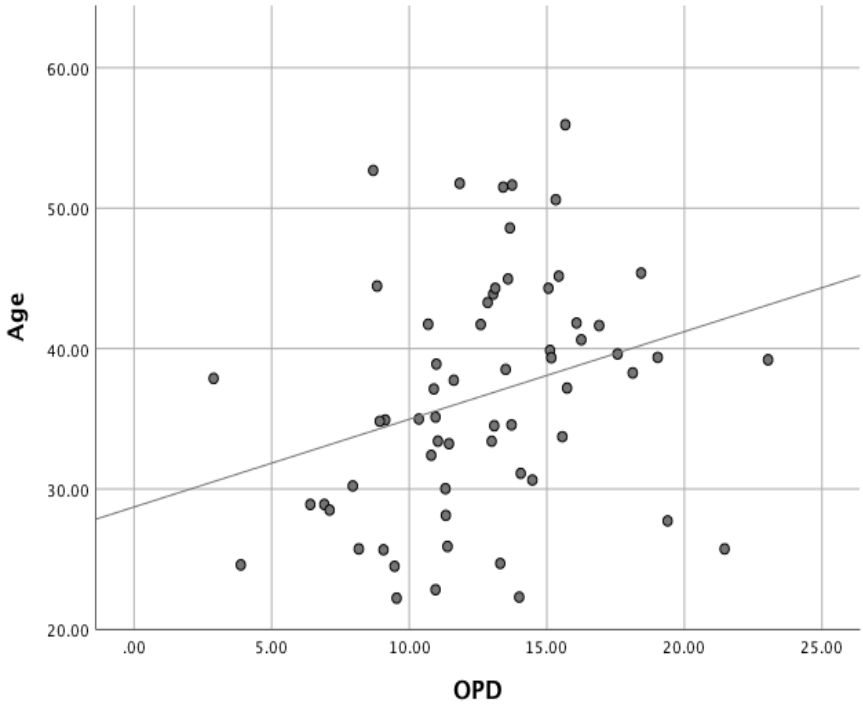


Figure 5.7. Scatterplot showing the relationship between adult age (years) and OPD

5.4.2. CORRELATIONS WITH AGE AND SUBADULT HISTOLOGY VARIABLES

The Pearson Correlation Coefficient showed a significant positive relationship with age and On.Ar ($r(82) = .482$, $p < .001$), On.Cr ($r(84) = .243$, $p = .02$), Ha.Ar ($r(80) = .232$, $p = .04$), and Po.Ar ($r(68) = .285$, $p = .02$) (Table 5.2). The increase in these variables with age is expected, however, the Pearson r value indicates that age may not be the sole influence for any of them. Neither Ha.Cr, OPD, %Ct.Ar, %Po.Ar correlated with age as expected in this sample.

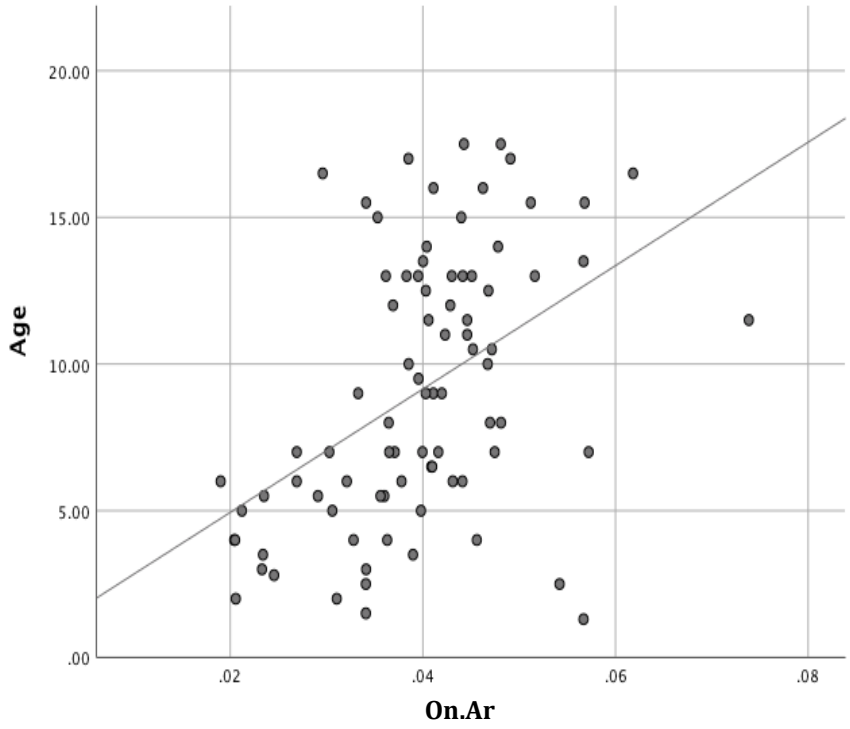


Figure 5.8. Scatterplot showing the relationship between subadult age (years) and On.Ar (r =.482)

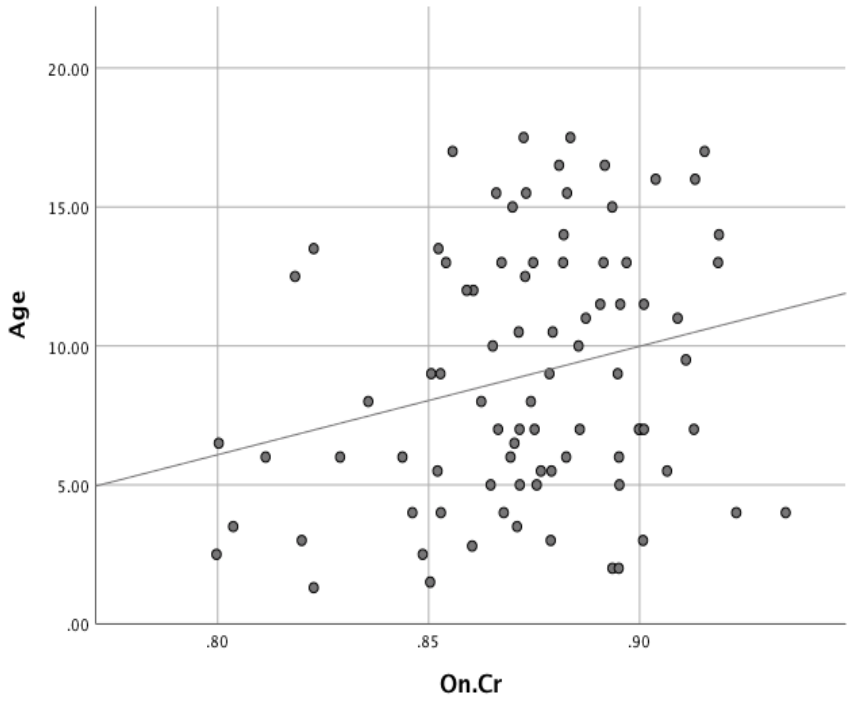


Figure 5.9. Scatterplot showing the relationship between subadult age (years) and On.Cr (r=.243)

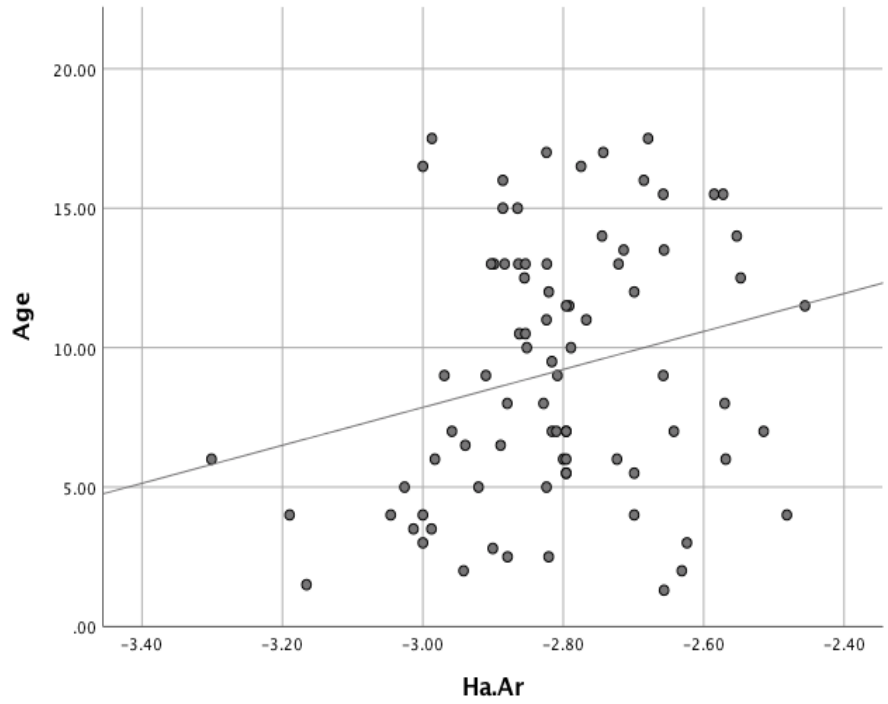


Figure 5.10. Scatterplot showing the relationship between subadult age (years) and Ha.Ar (r=.232)

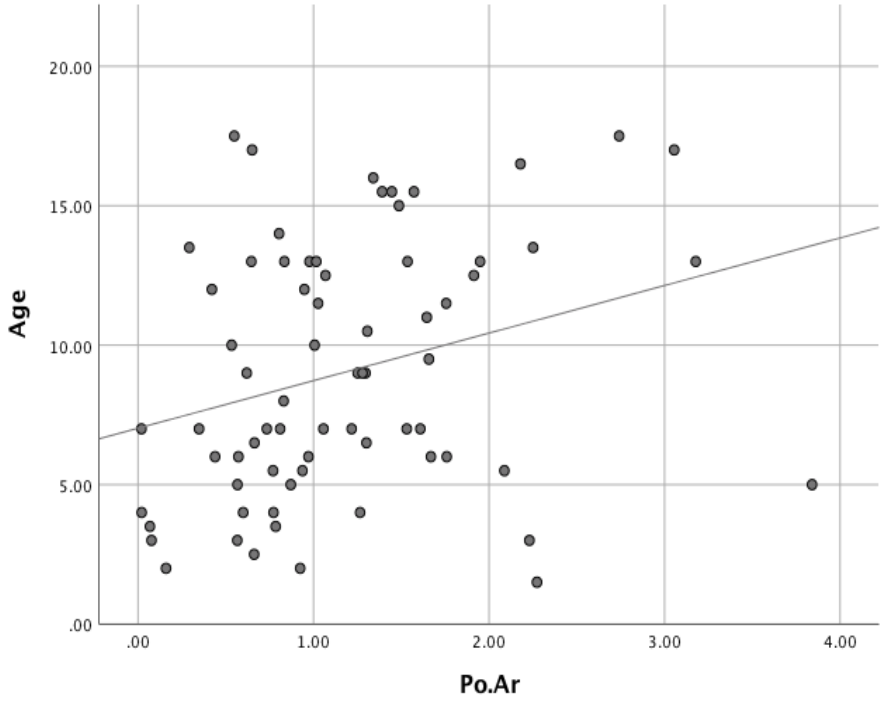


Figure 5.11. Scatter plots showing the relationships between subadult age (years) and Po.Ar (r=.285)

5.5. RESULTS FOR THE INVESTIGATION OF RESEARCH QUESTION 1: *Is there a difference in the rib histomorphometry of individuals that display lesions indicative of disease and individuals without lesions?*

The first question was explored by determining if there are differences in bone remodeling for each disease type. Then, an ANCOVA was used for each variable to determine if there is a relationship between disease and histomorphometry when age is included as a covariate. The results are presented here with adults and subadults analyzed in separate cohorts due to the known association between age and bone histomorphometry.

5.5.1. DIFFERENCES IN HISTOMORPHOMETRY OF DISEASE TYPES IN THE ADULT COHORT

Means for each histomorphometric variable within the disease type categories is presented in Table 5.3. Overall, there were few histomorphometric differences between disease types but the bone histology of individuals that showed evidence of metabolic and infectious disease appeared to be the least affected by disease processes. The means for each disease type are listed in Table 5.3 and bar graphs showing comparisons between disease types are shown in Figures 5.12a and 5.12b.

Table 5.3. Histomorphometric means for each category of adult disease type (On.Ar and Ha.Ar values have been normalized and are not the actual values (see Appendix 1.3))

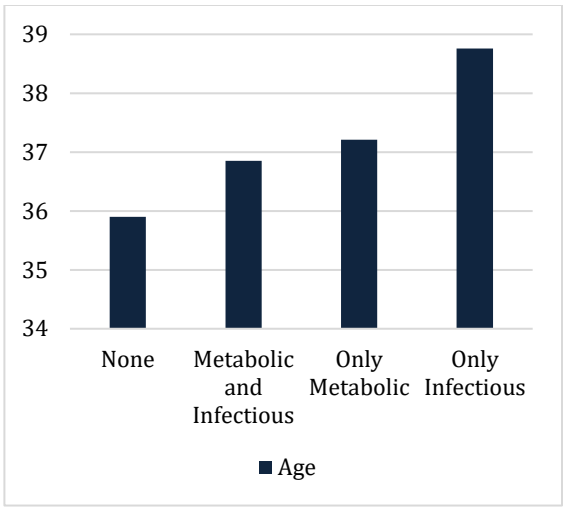
		Age	On.Ar	On.Cr	Ha.Ar	Ha.Cr	Po.Ar	%Po.Ar	%Ct.Ar	OPD	DZ
None	Mean	35.90	-1.41	.88	-2.86	.92	.69	3.88	40.35	12.41	3.92
	N	35	35	35	35	35	16	13	16	15	13
	SD	9.01	.13	.03	.017	.03	.10	1.89	10.91	5.26	2.93
Metabolic and Infectious	Mean	36.85	-1.44	.89	-2.88	.93	.42	2.17	38.02	13.07	3.88
	N	12	12	12	12	12	6	5	6	8	8
	SD	5.94	.08	.02	.10	.03	.10	.65	11.51	3.53	4.09
Only Metabolic	Mean	37.21	-1.42	.89	-2.85	.93	.79	3.19	39.33	12.74	3.89
	N	43	43	43	43	43	27	26	32	32	28
	SD	8.64	.12	.02	.14	.03	.59	2.17	10.39	2.97	2.57
Only Infectious	Mean	38.76	-1.40	.89	-2.89	.93	.59	3.03	41.95	12.68	2.88
	N	9	9	9	9	9	7	6	6	7	8
	SD	7.26	.07	.03	.28	.03	.32	2.11	12.17	5.27	2.85
Total	Mean	36.85	-1.42	.89	-2.86	.93	.70	3.25	39.73	12.70	3.75
	N	99	99	99	99	99	56	50	60	62	57
	SD	8.32	.11	.02	.16	.03	.48	2.00	10.57	3.88	2.87

Table 5.4. Means for adults with and without infectious diseases (On.Ar and Ha.Ar values have been normalized and are not the actual values (see Appendix 1.3))

Infectious disease		Age	On.Ar	On.Cr	Ha.Ar	Ha.Cr	Po.Ar	%Po.Ar	%Ct.Ar	OPD	DZ
Not Present	Mean	36.63	-1.41	.88	-2.85	.92	.75	3.42	39.67	12.63	4.98
	N	78	78	78	78	78	43	39	48	47	43
	SD	.12	.02	.15	.03	.03	.52	2.08	10.46	3.79	4.50
Present	Mean	37.67	-1.42	.89	-2.89	.93	.51	2.64	39.98	12.89	3.38
	N	21	21	21	21	21	13	11	12	15	16
	SD	.08	.02	.19	.03	.03	.25	1.61	11.48	4.27	3.44
Total	Mean	36.85	-1.42	.89	-2.86	.93	.70	3.25	39.73	12.70	4.54
	N	99	99	99	99	99	56	50	60	62	59
	SD	.11	.02	.16	.16	.03	.48	2.00	10.57	3.88	4.27

Table 5.5. Means for adults with and without metabolic diseases (On.Ar and Ha.Ar values have been normalized and are not the actual values (see Appendix 1.4))

Metabolic disease		Age	On.Ar	On.Cr	Ha.Ar	Ha.Cr	Po.Ar	%Po.Ar	%Ct.Ar	OPD	DZ
Not Present	Mean	36.49	-1.41	.88	-2.87	.92	.66	3.61	40.79	12.5	3.95
	N	44	44	44	44	44	23	19	22	22	22
	SD	.11	.03	.03	.19	.03	.37	1.94	5.14	5.14	3.46
Present	Mean	37.13	-1.43	.89	-2.85	.93	.73	3.03	39.12	12.81	4.89
	N	55	55	55	55	55	33	31	38	40	37
	SD	.11	.02	.02	.13	.03	.54	2.03	10.42	3.04	4.70
Total	Mean	36.84	-1.42	.89	-2.86	.93	.70	3.25	39.73	12.70	4.54
	N	99	99	99	99	99	56	50	60	62	59
	SD	.11	.02	.02	.16	.03	.48	2.00	10.57	3.89	4.28



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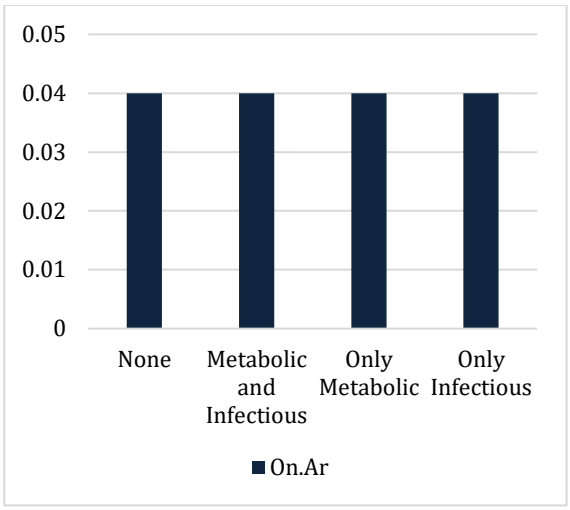

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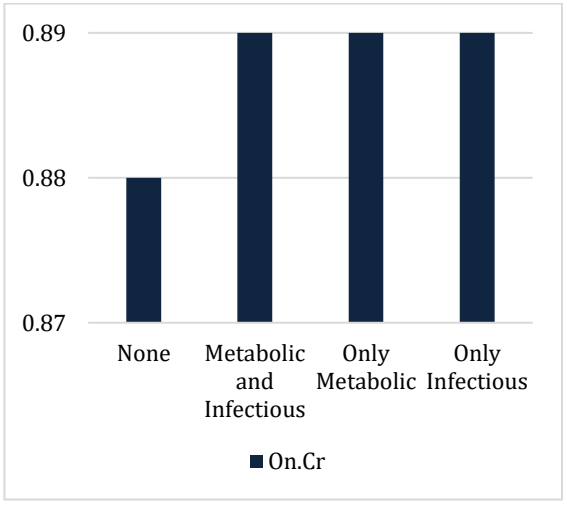

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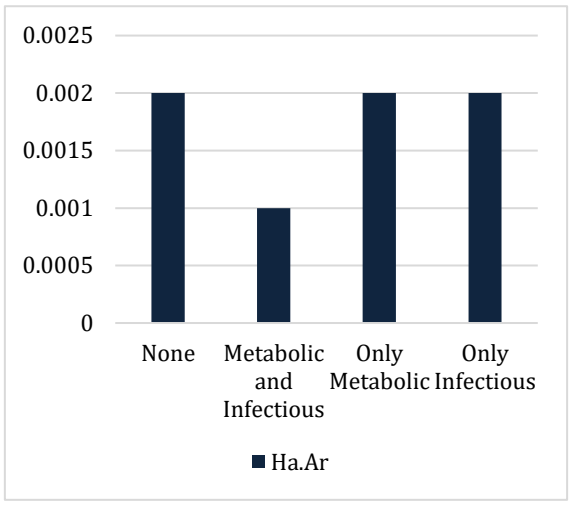

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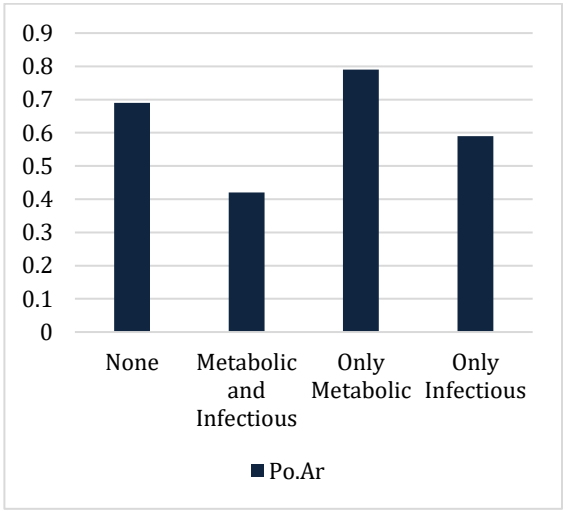

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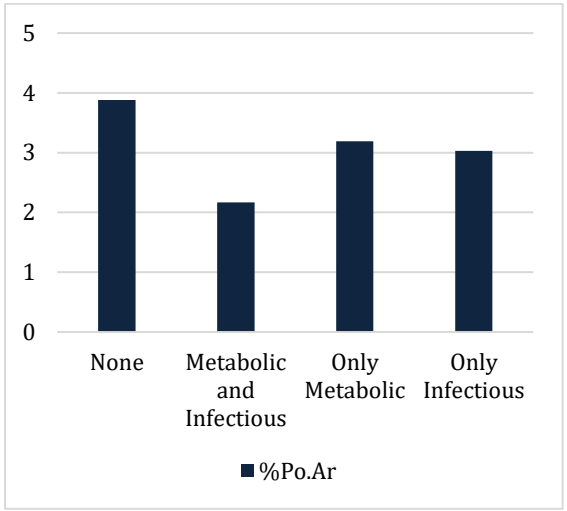
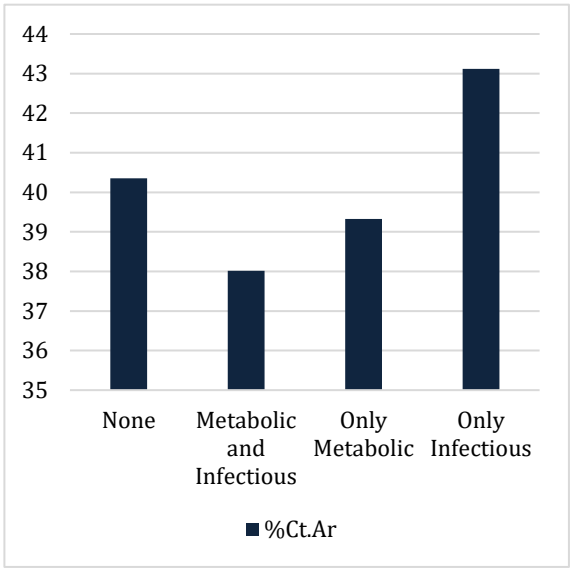


F.

Figure 5.12a. Adult mean histomorphometric variables for each disease type: A) On.Ar; B) On.Cr; C) Ha.Ar; D) Ha.Cr; E) Po.Ar; F) %Po.Ar



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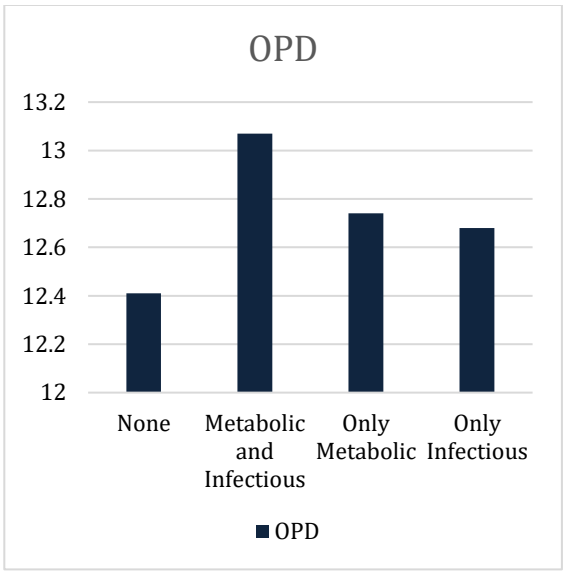

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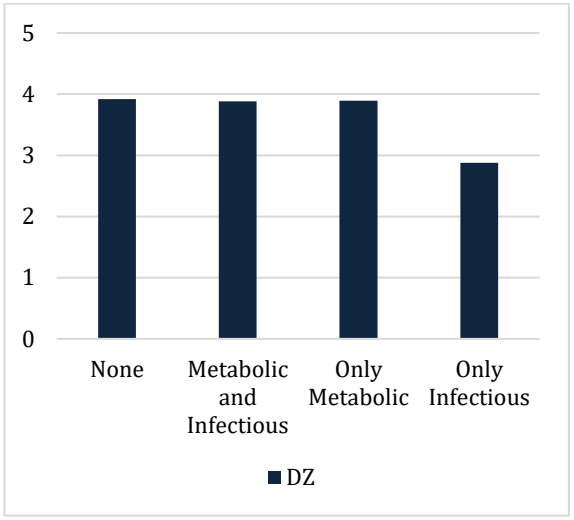

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Figure 5.12b. Adult mean histomorphometric variables for each disease type: G) %Ct.Ar; H) OPD; and I) DZ

5.5.1.1. Differences in histology between disease types in the adult cohort

Null hypothesis 1.1 was not rejected because there were no differences between those who have evidence of only metabolic disease and those of other disease types. Null hypothesis 1.2 was rejected because adults who suffered from infectious disease (infectious disease group and metabolic and infectious disease group) had significantly less porous cortices than those without evidence of infectious disease ($t(43.53)=2.29$, $p=.03$) (Table 5.6). Finally, null hypotheses 1.3 was rejected because adults who suffered from comorbidities of both metabolic and infectious disease had significantly less porosity area than those who only show evidence of metabolic disease ($t(30.56)=-3.07$, $p=.004$) (Table 5.7). Additionally, those with comorbidities have significantly smaller amount of porosity ($t(18.79)=2.34$, $p=.03$) and lower percent porosity area ($t(16)=2.85$, $p=.01$) than those who do not have any lesions (Table 5.8), so null hypothesis 1.4 was also rejected.

Table 5.6. Comparison of total Po.Ar between adult individuals who suffered from infectious disease and those without evidence of infectious disease ($p=.03$)

	Disease Type	*n*	Mean	SD
Po.Ar (mm^2)	Metabolic and Infectious	6	.43	.10
	No lesions	16	.69	.40
%Po.Ar	Metabolic and Infectious	5	2.17	.65
	No lesions	13	3.89	1.89

Table 5.7. Comparison of total Po.Ar between adult individuals who suffered from metabolic and infectious disease and those that only show evidence of metabolic disease ($p=.003$)

	Infectious Disease	*n*	Mean (mm^2)	SD
Po.Ar	Not Present	43	.75	.52
	Present	13	.51	.25

Table 5.8. Comparison of total Po.Ar ($p=.03$) and %Po.Ar ($p=01$) between adult individuals who suffered from metabolic and infectious disease and those without lesions indicative of disease

	Disease Type	*n*	Mean (mm^2)	SD
Po.Ar	Metabolic and Infectious	6	.43	.10
	Only Metabolic	27	.79	.60

Differences between individuals with scurvy were first examined in the group with only metabolic disease to control for effects due to infection. Then, scurvy was compared in the

metabolic and infectious disease group, finally both groups were combined to look at the overall differences between those with and without evidence of scurvy. Scurvy is present in 74.4% (32/43) of those with only metabolic disease. When adults with evidence of scurvy in the metabolic group are compared to those without scurvy, the scorbutic individuals have significantly lower percent cortical area ($t(30)=2.70$, $p=.01$) (sexes combined), smaller osteon area ($t(41)=2.67$, $p=.01$), and more circular Haversian canals ($t(41)=-2.56$, $p=.01$) (Table 5.9). In the metabolic and infectious disease group, scurvy is present in 83.3% (10/12) of individuals with comorbidities. Those with scurvy showed lower percent cortical area than those without scurvy ($t(4)=3.18$ $p=.03$); however, the sample size is very small for individuals with cortical area data in this group ($n=6$) (Table 5.10). When all disease types are combined, the data showed similar results as the previous comparisons with those who have evidence of scurvy showing lower percent cortical area ($t(59)=2.84$, $p=.006$) (sexes combined) and greater Haversian canal circularity ($t(97)=-2.02$, $p=.05$) than those without evidence of scurvy (Table 5.11).

Table 5.9. Comparisons of %Ct.Ar (sexes combined) ($p=.01$), On.Ar ($p=.01$), and Ha.Cr ($p=.01$) between adults from the metabolic disease group who suffered from scurvy and those without evidence of scurvy (On.Ar values are normalized and not reflective of actual values (see Appendix 1.3))

	Scurvy	*n*	Mean	SD
%Ct.Ar	Not present	7	47.88	10.39
	Present	25	36.93	9.23
On.Ar (mm^2)	Not present	11	-1.34	.09
	Present	32	-1.44	.11
Ha.Cr (mm^2)	Not present	11	.91	.02
	Present	32	.93	.02

Table 5.10. Comparison of %Ct.Ar (sexes combined) ($p=.03$) between adults from the metabolic and infectious disease group and who suffered from scurvy and those without evidence of scurvy.

	Scurvy	*n*	Mean (mm^2)	SD
%Ct.Ar	Not present	2	50.60	10.54
	Present	4	31.74	5.05

Table 5.11. Comparisons of %Ct.Ar (sexes combined) (*p*=.006) and Ha.Ar (*p*=.05) between adults who suffered from scurvy and those without evidence of scurvy (Ha.Ar values are normalized and not reflective of actual values (see Appendix 1.3))

	Scurvy	*n*	Mean	SD
%Ct.Ar	Not present	31	43.47	11.07
	Present	30	36.21	8.73
Ha.Cr (mm^2)	Not present	56	.92	.03
	Present	43	.93	.02

5.5.1.2. Analysis of covariance with age in the adult cohort

While not all histomorphometric variables in this sample showed a significant correlation with biological age, it is widely accepted that age is a primary contributor to the variation present in rib histomorphometry (Dominguez and Agnew 2016; Stout and Paine 1992). A test for homogeneity of regression slopes showed that disease and age are independent from one another, so age was treated as a covariate in a one-way Analysis of Covariance (ANCOVA) test for the five variables with which it had a relationship (On.Ar, On.Cr, Ha.Cr, %Ct.Ar, and OPD). However, the ANCOVA showed no significant relationship with disease. Since some variables that were expected to show a relationship with age did not, an experimental one-way ANCOVA was performed where disease was treated as a covariate instead of age for all variables. In this test, both Po.Ar ($F(52, 2) = 38.50$, $p = .03$) and %Po.Ar ($F(46, 2) = 25.60$, $p = .04$) showed a strong relationship with age. Both variables showed a partial Eta above .99, indicating disease plays a large role in the degree of porosity for the adult cohort.

5.5.2. DIFFERENCES IN HISTOMORPHOMETRY OF DISEASE TYPES IN THE SUBADULT COHORT

The means for each disease type are listed in Tables 5.12 and 5.13, and graphs showing comparisons between disease types are shown in Figure 5.12A-H. There were few statistically significant histomorphometric differences between disease types in the subadult cohort. Overall, individuals with osseous pathological changes indicative of metabolic and infectious disease appear to have less porotic ribs while those without lesions show the greatest bone loss, suggesting those with metabolic and infectious disease were better at maintaining bone.

Table 5.12. Histomorphometric means for each category of subadult disease type (On.Ar and Ha.Ar values have been normalized and are not the actual values (see Appendix 1.4))

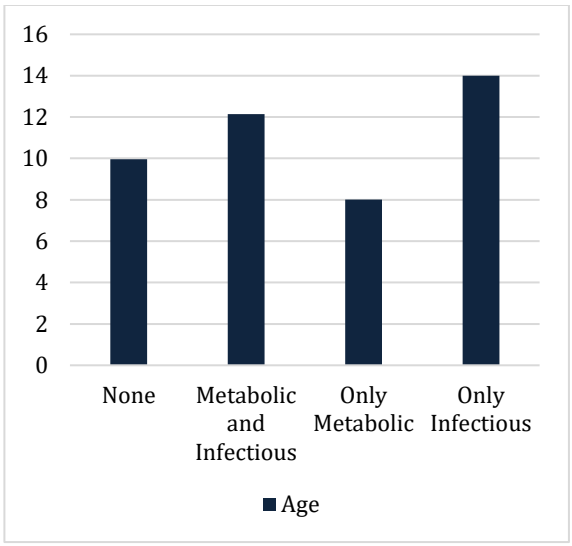
		Age	On.Ar	On.Cr	Ha.Ar	Ha.Cr	Po.Ar	%Po.Ar	%Ct.Ar	OPD
None	Mean	9.96	-1.39	.87	-2.79	.91	1.41	6.93	52.73	2.15
	N	15	15	15	15	15	12	13	13	13
	SD	3.69	.07	0.28	.10	.03	.54	2.24	11.63	.99
Metabolic and Infectious	Mean	12.14	-1.38	.87	-2.86	.90	.78	4.63	57.67	2.89
	N	7	7	7	7	7	6	5	4	5
	SD	2.54	.01	.02	.11	.03	.38	.78	2.49	1.27
Only Metabolic	Mean	8.01	-1.43	.87	-2.81	.91	1.17	6.62	47.54	1.88
	N	62	59	61	57	60	51	45	45	46
	SD	4.77	.13	.03	.17	.03	.83	3.85	8.19	1.04
Only Infectious	Mean	14.00	-1.35	.89	-2.75	.92	1.45	3.54	52.52	1.11
	N	3	3	3	3	3	1	2	2	2
	SD	2.18	.10	.02	.16	.04	.	.51	8.91	.49
Total	Mean	8.89	-1.42	.87	-2.81	.91	1.19	6.43	49.38	1.98
	N	87	84	86	82	70	65	64	64	66
	SD	4.63	.12	.03	.16	.03	.76	3.43	9.14	1.06

Table 5.15. Means for subadults with and without infectious diseases (On.Ar and Ha.Ar values have been normalized and are not the actual values (see Appendix 1.4))

Infectious disease		Age	On.Ar	On.Cr	Ha.Ar	Ha.Cr	Po.Ar	%Po.Ar	%Ct.Ar	OPD
Not Present	Mean	8.40	-1.42	.87	-2.81	.91	1.21	6.69	48.71	1.94
	N	77	74	76	72	75	63	58	58	59
	SD	6.62	.12	.03	.03	.03	.78	3.54	9.22	1.03
Present	Mean	12.70	-1.37	.87	-2.83	.91	0.87	4.32	55.95	2.38
	N	10	10	10	10	10	7	7	6	7
	SD	2.49	.09	.02	.13	.03	.42	.85	5.16	1.36
Total	Mean	8.89	-1.42	.87	-2.81	.91	1.18	6.43	49.38	1.98
	N	87	84	86	82	85	70	65	64	66
	SD	4.63	.12	.03	.16	.03	.76	3.43	9.14	1.06

Table 5.16. Means for subadults with and without infectious diseases (On.Ar and Ha.Ar values have been normalized and are not the actual values (see Appendix 1.4))

Metabolic disease		Age	On.Ar	On.Cr	Ha.Ar	Ha.Cr	Po.Ar	%Po.Ar	%Ct.Ar	OPD
Not Present	Mean	10.34	-1.39	.87	-2.80	.91	1.41	6.66	53.16	2.05
	N	17	17	17	17	17	12	14	14	114
	SD	3.67	.07	.03	.10	.54	.54	2.37	11.30	1.02
Present	Mean	8.53	-1.42	.87	-2.82	.91	1.13	6.37	48.33	1.96
	N	70	67	69	65	58	58	51	50	52
	SD	4.78	.13	.03	.17	.79	.79	3.68	8.27	1.08
Total	Mean	8.89	-1.42	.87	-2.81	.91	1.18	6.43	49.38	1.98
	N	87	84	86	82	85	70	65	64	66
	SD	.12	.12	.03	.16	.03	.76	3.43	9.14	1.06



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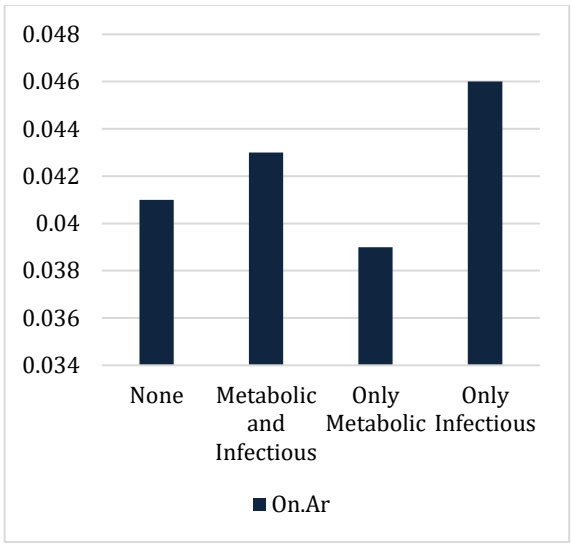

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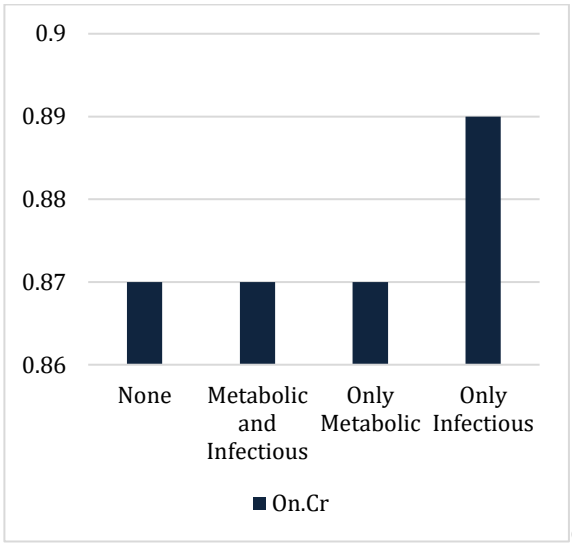

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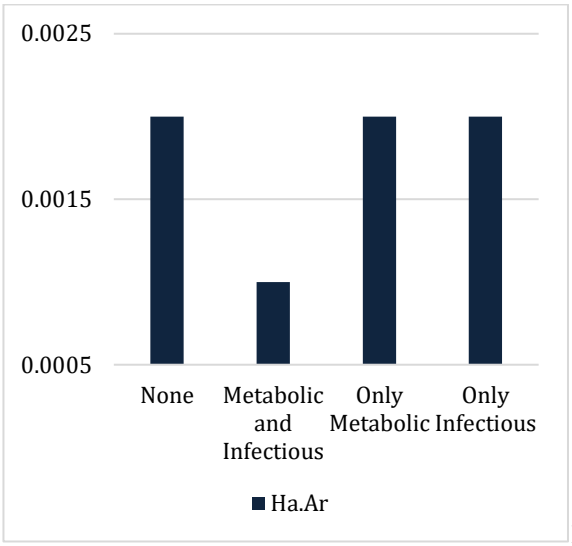

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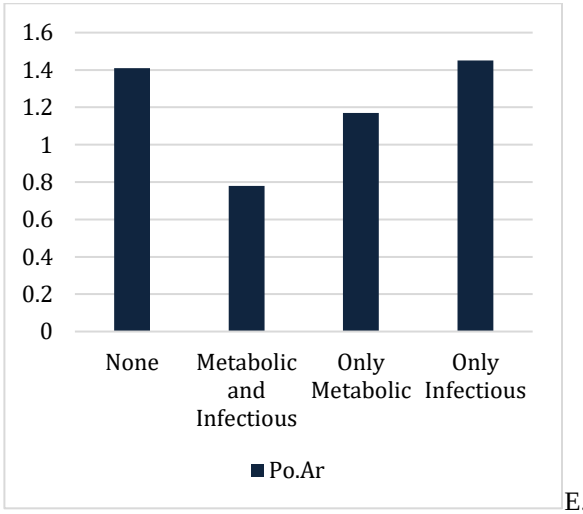

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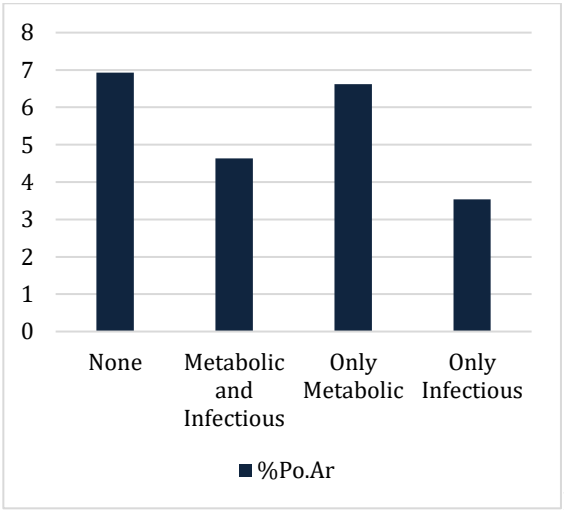
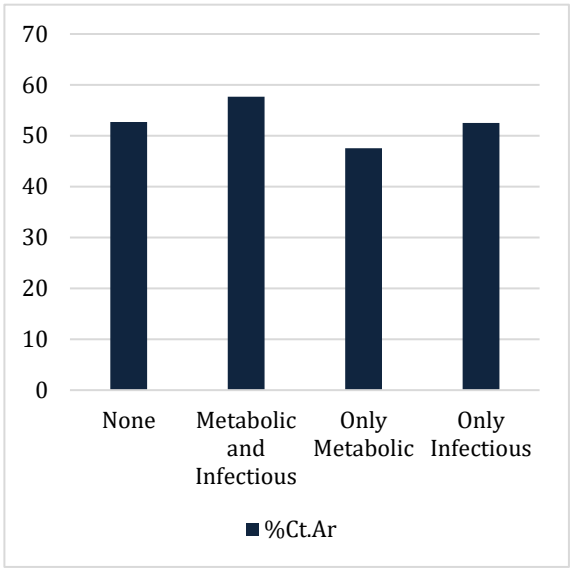


F.

Figure 5.13a. Bar graphs showing subadult mean histomorphometric variables for each disease type: A) Age; B) On.Ar; C) On.Cr; D) Ha.Ar; E) Ha.Ar; F) Po.Ar



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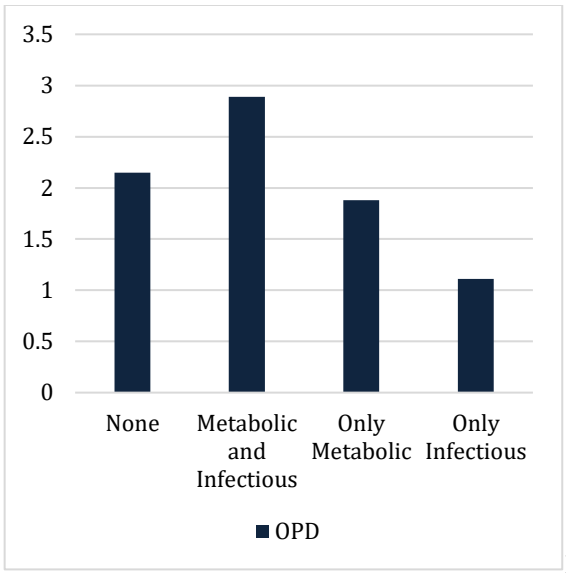

I.

Figure 5.13b. Bar graphs showing subadult mean histomorphometric variables for each disease type: G) %Po.Ar; H) %Ct.Ar; I) OPD

5.5.2.1. Differences in bone histology variables between disease types in the subadult cohort

Every null hypothesis was rejected for the subadult cohort. Null hypothesis 1.3 was rejected because individuals with lesions only indicative of metabolic disease showed significantly lower %Ct.Ar ($t(47)=2.44$, $p=.02$) and lower OPD ($t(49)=2.02$, $p=.05$) (Table 5.17) than those who have metabolic and infectious comorbidities. The second null hypothesis was rejected because individuals who have lesions indicative of infectious disease showed significantly less %Po.Ar in the rib cortex than those who do not have lesions indicative of infectious disease ($t(63)=1.75$, $p<.001$) (Table 5.18). Finally, the third and fourth null hypotheses were rejected because individuals who have lesions indicative of both metabolic and infectious disease show significantly lower Po.Ar than individuals without lesions ($t(20)= 2.56$, $p=.02$) (Table 5.19). In each of these cases, subadults with infectious disease and with comorbidities of infectious and metabolic disease seem to be experiencing more balanced bone remodeling than those with only metabolic disease and those without lesions.

When subadults with and without evidence of scurvy are compared within the metabolic disease group, scorbutic children show significantly smaller osteon area ($t(57)=2.08$, $p=.05$) and when groups are combined those with scurvy show significantly smaller osteon area ($t(82)=1.94$, $p=.02$) (Table 5.20). All subadults in the metabolic and infectious disease group show signs of scurvy, so this was group could not be tested.

Table 5.17. Difference in %Ct.Ar (*p*=.02) and OPD (*p*=.05) between subadults who suffered from metabolic and infectious disease and those with lesions only indicative of metabolic disease

	Infectious Disease	*n*	Mean (mm^2)	SD
%Po.Ar	Not present	58	6.69	3.54
	Present	7	4.32	.85

Table 5.18. Difference in %Po.Ar between subadults who suffered from infectious disease and those without lesions indicative of infectious disease (*p*=.0002)

	Disease Type	*n*	Mean (mm^2)	SD
Po.Ar	Metabolic and Infectious	6	1.41	.54
	No Lesions	12	.78	.37

Table 5.19. Difference in total Po.Ar between subadults who suffered from metabolic and infectious disease and those without lesions indicative of disease (*p*=.01)

	Disease Type	*n*	Mean (mm^2)	SD
%Ct.Ar	Metabolic and Infectious	4	57.67	2.48
	Only Metabolic	45	47.54	8.19
OPD	Metabolic and Infectious	5	2.88	1.26
	Only Metabolic	46	1.88	1.04

Table 5.20. Differences in On.Ar between subadults that show evidence of scurvy and those that do not have lesions indicative of scurvy in the only metabolic group (*p*=.05) and with all disease types combined (*p*=.02). On.Ar values have been normalized and are not the actual osteon size values (see Appendix 1.4)

On.Ar	Scurvy	*n*	Mean (mm^2)	SD
Only Metabolic	Not present	4	-1.31	.12
	Present	55	-1.44	.12
All disease types	Not Present	21	-1.37	.01
	Present	63	-1.43	.12

5.5.2.2. Analysis of covariance with age in the subadult cohort

Since On.Ar, Ha.Ar, and Po.Ar all showed correlations with age in the subadult cohort, disease comparisons for these variables were conducted in a one-way ANCOVA where age could be controlled as a covariate contributing to the variance in histomorphometry. After controlling for age, the ANCOVA showed no significant differences between histomorphometry and disease groups for any variable in the subadult cohort. While the sample size is too low to determine statistical significance for the infectious group, these children showed the highest mean age with the smallest and most circular osteons and Haversian canals, the highest porosity area but lowest percent porosity area, and the lowest OPD (Table 5.15). There were no

statistically significant differences between individuals with scurvy and those without for any of the histomorphometric variables as determined by the one-way ANCOVA.

5.6. RESULTS FOR THE INVESTIGATION OF RESEARCH QUESTION 2

The second question was explored using Pearson's correlation coefficient to determine if there is a correlation between stable isotope values indicative of diet ($\delta^{13}C$) and nutritional deficiency ($\delta^{15}N$) with bone remodeling. Then, an ANOVA and an ANCOVA were performed on the significant variables to determine how much of the variation present in the dependent variable could be attributed to the change in isotope values without age as a covariate and with age as a covariate. The results are presented here with adults and subadults presented as separate cohorts due to the association between age and bone histomorphometry.

5.6.1. COMPARISONS BETWEEN STABLE ISOTOPES AND BONE HISTOLOGY IN THE ADULT COHORT

Osteon area showed a significant positive linear relationship with $\delta^{15}N$ isotope values ($r(15)=.564$, $p = .02$) where individuals with higher $\delta^{15}N$ value have larger osteons; however, the adjusted R_2 value is low, indicating that $\delta^{15}N$ may not contribute much to the variation observed in osteon area. To determine how much of the variation in On.Ar could be attributed to $\delta^{15}N$, an ANOVA was performed. The analysis showed $\delta^{15}N$ is significantly associated with On.Ar ($F(10, 6)= 8.08$, $p=.009$) (Figure 5.14) where the partial Eta is .931, indicating much of the variation present in osteon area can be attributed to the change in $\delta^{15}N$ values.

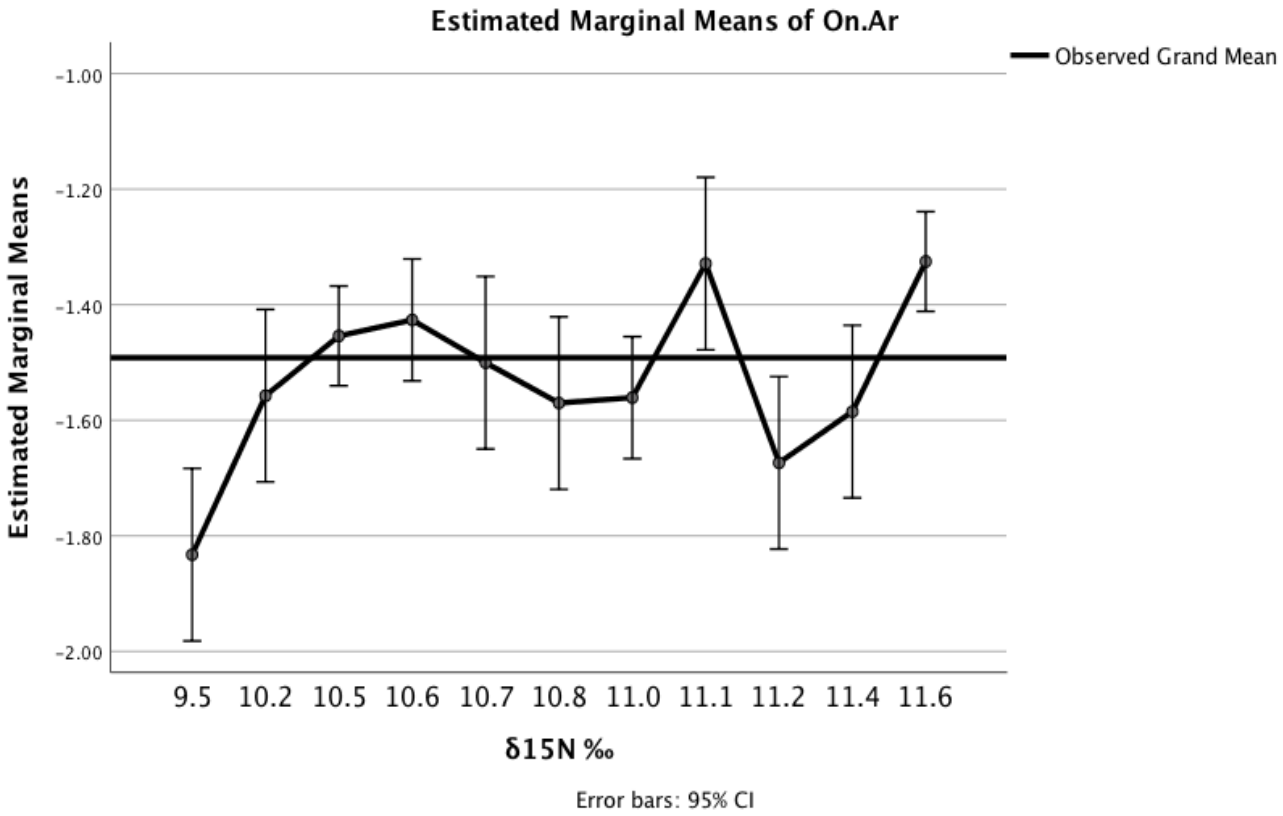

Figure 5.14. The $\delta^{15}N$ isotope values show a significant positive linear relationship with osteon area (p=.009). On.Ar values have been normalized and are not the actual osteon size values (see Appendix 1.3)

Since a correlation between age and On.Ar was observed in the adult cohort, an ANCOVA was performed to determine if the relationship between On.Ar and $\delta^{15}N$ changes when age is included as a covariate. The results of the ANCOVA show that $\delta^{15}N$ has a significant relationship with osteon area ($F(10, 5)= 6.63$, $p= .05$) where $\delta^{15}N$ accounts for at least 93% of the variation in osteon area (Figure 5.15). There were no significant correlations between histomorphometric variables and $\delta^{13}C$ isotope ratios, indicating that the remodeling process of the rib microstructure may not have had enough time before death to respond to the introduction of relief food in a way that could be measured by histomorphometry.

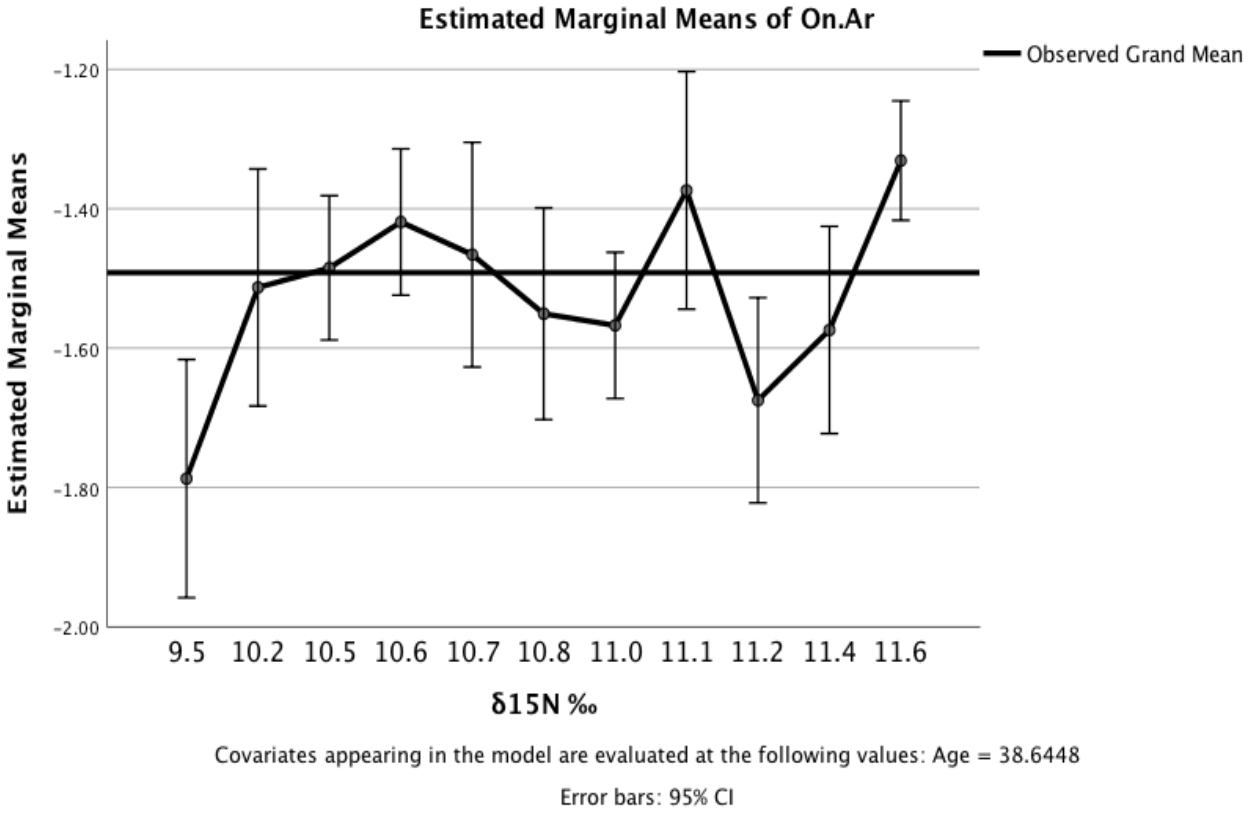

Figure 5.15. The $\delta^{15}N$ isotope values show a positive linear relationship with osteon area after controlling for age in the ANCOVA. The data now show a strong statistical relationship between $\delta^{15}N$ and osteon area ($p=.03$; $adjR^2=.838$). On.Ar values have been normalized and are not the actual osteon size values (see Appendix 1.3)

5.6.2. COMPARISONS BETWEEN STABLE ISOTOPES AND BONE HISTOLOGY IN THE SUBADULT COHORT

There were no correlations between $\delta^{15}N$ isotope ratios and histomorphometric variables in the subadult cohort. However, the sample size for subadults with both isotope data and histomorphometric data was small (n=8). Adding to the issue of small sample size among the subadult sample, these children, due to their young age, have few secondary osteons and Haversian canals, limiting the amount of variation that can be observed in the sample.

A significant positive linear relationship was observed between Haversian canal area and $\delta^{13}C$ isotope ratios ($r(6)$ =.727, p=.05); indicating that the association between $\delta^{13}C$ and Haversian canal area is strong. However, in the previous section it was determined that age and Ha.Ar are correlated for subadults in this sample. Unfortunately, an ANOVA could not be performed on the subadult sample to determine the strength of the relationship between Ha.Ar and $\delta^{13}C$ when age is controlled for due to the unreliability of outcomes with small sample sizes.

CHAPTER 6. DISCUSSION

This chapter will discuss the results reported in Chapter 5 to better understand how disease and diet during the Great Famine influenced histological variation in the sample from the Kilkenny Union Workhouse. In this section, each research question will be addressed and discussed in the order it was presented in the introduction to this thesis and historical context will be provided to tease out how the results may reflect variation in frailty in the sample. Additionally, a section on how the vitamin deficiency disease pellagra may have been misdiagnosed in the inmates of the Kilkenny Union Workhouse will be provided and finally, the role of structural violence played in the enormous loss of life during this time will be discussed.

As stated in Section 1.2., the aim of this thesis is to assess how disease and changes in diet due to structurally induced famine conditions affect rib bone microstructure. The differences observed in cortical rib bone loss (porosity) between disease types are likely representative of hidden social, genetic, and biological diversity among the inmates of the Kilkenny Union Workhouse. During the Great Famine, risk of death was highly correlated with social structures maintained by the Poor Laws that were set more than 100 years before the potato blight arrived (Smyth 2012). Type of homestead, form of employment, and lack of literacy of the English language all contributed to vulnerability during this catastrophic period (Smyth 2012).

On the surface, the victims of the Famine who lived and died in the workhouses may appear to be a homogeneous group of poor people who suffered equally regarding housing, single-food dependence, and religious and cultural discrimination, but the experience of the varied and individual circumstances contributed to their risk of death during the Famine. Among them are the conditions of early childhood such as a seasonal famine and type of housing environment. These factors can influence immune strength and adaptability in later life (Edwards 2019) and although these differences are difficult to recognize in the skeleton, they would have played a role in access to relief efforts such as the workhouses and the probability of survival if access was granted.

As described in Chapter 2, the Irish workhouses were designed based on a utilitarian method of construction designed by architect George Wilkinson (1814-1890) inspired by the style of

punishment often attributed to Jeremy Bentham (Bentham 1789) that aimed to deter the poor from seeking relief (Foucault 1975; Smyth 2012). Inspired by nineteenth century prisons, which deliberately created a divide between the law-abiding and the lawless people of the city, they were enclosed by tall stone walls with separate living quarters for men, women, and children (Smyth 2012). The poor in the workhouses were not convicted criminals, but historical accounts report they were treated as such by the British government (Smyth 2012). Nevertheless, the workhouses provided relief to an alternative life of starvation and homelessness.

Often when reading about the Great Famine and multiple times in this thesis, the victims of the Great Famine are referred to by their socio-economic class—the destitute, poor, paupers, or "poor devils" as Karl Marx (1818-1883) referred to them (Marx 1906: p.774; Clarkson and Crawford 2001; Murphy and Geber 2012; Geber 2015). However, being poor during a famine is very different from being poor at any other time and in nineteenth century Ireland living in poverty was not enough to grant access to these institutions. Requirements to enter a workhouse varied throughout the Famine years but from the beginning access was restricted by age, sex, family history, and income (Geber 2015). As described earlier, the poor law unions prioritized access to indoor relief, which included shelter and food (primarily imported North American maize), based on the perceived vulnerability of the person in need (Clark 2011). The elderly and children were prioritized most often, followed by women, particularly if they were pregnant or nursing. Young men were the least likely to be accepted into the workhouse except under dire circumstances (Clark 2011). This was likely to happen when the public works scheme ended, or the men were too sick to perform physical labor.

Later, when the Poor Law was amended, entry was restricted for tenant farmers and was based on the size of land they leased. Joel Mokyr, an economist who is credited with determining the most likely death toll of the Great Famine, estimated that income and type of housing played the greatest role in the increased risk of death for the Irish poor. By his estimate, for every one-pound increase in income per person, between 65,000 and 125,000 less people would have died (Mokyr 1983; Smyth 2012). Before the potato blight two-thirds of families were dependent on 57% of the potatoes consumed by humans in Ireland. Cottiers who lived in small, often one

room bothán consumed 13% of the total potatoes and would be the most desperate for relief when the crop was ruined (Smyth 2012).

Based on housing information gathered from both the 1841 and 1851 census, fourth class cottier housing was correlated with ~600,000 deaths. When the Gregory Clause required those who entered the workhouse to give up all but one quarter acre of their land and the Rate-in-Aid Act required "Irish property to pay for Irish poverty", many of these properties were destroyed by landowners who did not want to pay taxes on the holdings. The removal of homes can be observed statistically when the two census records are compared and show that of the 271,000 homesteads that were absent between the 1841 census and 1851 census—72.4% were cottier homes (Census of Ireland, 1841, 1851; Smyth 2012). In Kilkenny, 82-84% of these types of properties were removed, a dark representation of the number of people who died, were forcibly displaced, or made homeless during this time. Further, the 1851 census also records an increase in second- and first-class housing at 20.7% and 25.3%, respectively, which Smyth acknowledges as a demonstration of the change in social structure after the Famine (Smyth 2012).

Although the Famine did primarily affect the lowest classes, as the potato crop continued to fail for multiple seasons third-class tenant farmers who could not afford to emigrate were forced to give up their land (Smyth 2012). As a result, a hole was created in the socioeconomic ladder causing a greater divide between the upper and lower classes and leveling the lowest classes into a single floor of poverty. This is not to say there was never assistance from higher classes. Many landlords offered to pay for their tenants' emigration as it was cheaper than paying the rate on their properties and members of various levels of society contributed to the well-being of the people in the workhouses including clergymen, medical professionals, and the workhouse officials (Smyth 2012). Unfortunately, many of them succumbed to typhus while working to help the poor.

As a result of the continued failure of the potato crop and the gaps in public works relief access, the workhouse environment was composed of multiple classes of people who regularly lived and interacted with one another to create a composite of individuals with varying economic

histories, backgrounds, and innate resilience—particularly in the later years of the Famine when the burial location in Kilkenny began to be used. While it is unlikely the clergy, medical professionals, or workhouse officials would have been buried on the workhouse grounds, it is not entirely impossible, and this too would contribute to the variation in frailty among the people buried on the grounds of the Kilkenny Union Workhouse.

6.1 SEX DIFFERENCES IN HISTOMORPHOMETRY

In the Kilkenny Union Workhouse sample, adult females exhibited greater percent cortical area when compared to adult males in this study, which is a pattern not often observed in bone histology studies. It is more common that there are no differences in cortical area in the rib between the sexes (Cho et al. 2002; Mulhern 2000; Stout 1986). While females and males had nearly the same size pores, females had greater percent porosity within their cortical bone. These results, though not statistically significant, are consistent with other studies that show females exhibit greater porosity than males (Ericksen 1991; Thompson 1979), however bone loss such as this usually occurs in the femur and is often linked to endocrine changes during pregnancy or lactation. Cortical porosity in older women is also frequently link to age, where women are more likely to exhibit osteoporosis (Ahmed et al. 2015; Bala, Zebaze, and Seeman 2015). Within the Kilkenny Union Workhouse women and men, while separated, were provided with similar allocations of relief food (Geber 2015). However, Geber mentions males may have required more nutrients than females due to differences in biological metabolism making the male ration inadequate to account for these differences (Geber 2015). An alternative explanation is that the larger cortices in females is representative of the female buffering hypothesis (Stinson 1985). This hypothesis suggests that due to reproductive demands, women are biologically resilient or buffered against stress so that when compared to their male counterparts males appear to be more sensitive to stressors (Ruff, 2007; Cohen and Crane-Kramer 2007). Since this population experienced regular seasonal famines, it is possible the females were buffered to better manage their calcium stores and maintain balanced bone remodeling than males.

While only percent cortical area was significant between sexes, to explore whether age may have contributed to recorded lower bone density in adult females, the sample was divided into

a younger (18-35 years; $\overline{x}$ =29.9 ±4.43) (n=49; females=31; males=18) and an older cohort (36+ years; $\overline{x}$ =43.6 ±4.95) (n=50; females=24; males=26). Younger males had significantly lower percent cortical area ($t(65) = 3.14$, $p = .003$) (males M=35.99, SD =9.42; females M=43.91, SD=10.67), greater porosity area (means), and greater percent porosity area (means) than younger females indicating that younger males were losing more bone than younger females. While remodeling in the rib is known to less likely be affected by mechanical loading, it is possible that greater loads suffered during labor performed in public works caused an increase in remodeling that could not be compensated for due to a lack of dietary resources. As expected, older females have greater porosity area ($t(27) = 2.20$, $p = .04$) (females M= .87, SD =.42; males $\overline{x}$ =.55, SD =.35) and greater percent porosity ($t(22) = 4.60$, $p = .0001$) (females M =4.96, SD =.1.72; males M= 2.21, SD =1.06), but they also have greater cortical area than older males ($t(27) = 2.50$, $p = .02$) (females M=39.24, SD =6.69; males M=31.89, SD =9.16).

Interestingly, a notable difference was present between younger and older men in the adult cohort. These results indicate younger males ($\overline{x}$ =29.45, ±4.64 years) have less bone density than older men ($\overline{x}$ =44.16, ±4.38 years) (M= 3.68 SD= 2.17; M= 2.21, SD= 1.06), which is not expected since older individuals are known to have more porous and thinner cortices than younger people. Older males have greater OPD, which is consistent with studies that show osteon accumulation over time, though this difference is small. According to the poor law requirements, older males were more likely to be allowed in the workhouse because they may have been deemed not fit for public works. There they would have received relief food which may have allowed them to maintain bone remodeling at a more consistent rate than the younger men who were put to hard labor without sufficient nutritional intake to replace the calcium lost through osteoclastic resorption. Older males and younger females may also have suffered more from infectious disease than metabolic disease in the crowded workhouses than younger males who were less likely to contract infectious diseases in an outdoor environment. This is consistent with the data found in Question 1 and will be discussed below. The differences in bone loss between the sexes are likely a function of combined cultural, biological, and disease experiences but they were also a function of structural violence since differential access to resources dictated the bone health of these people.

6.2. ADDRESSING QUESTION 1: HISTOMORPHOMETRIC COMPARISONS OF DISEASE TYPES

The first year the Kilkenny Union Workhouse burials were in use was 1847. This year is widely known as "Black 47", the deadliest year of the Great Famine. Between 1846 and 1847 around 400,000 people died (Kinealy 1997) of typhus and other infectious diseases with similar symptoms, often referred to as "famine fever" (Mokyr and Gráda 2002). During this time, more people died in County Kilkenny than any other year of the Famine (Cousens 1960). While it was reported that most deaths were due to infectious disease (Census of Ireland 1856), when Geber (2015) conducted paleopathological analysis on the Kilkenny Union Workhouse skeletons, they found evidence of infectious disease in only 15.8% of the sample, whereas more than half showed evidence of scurvy (51.4%), described in this thesis as a metabolic disease. The sample used for histological analysis is representative of the burial population in terms of age, sex, and disease distribution. The following section discusses the differences observed in the histomorphometry for adults and subadults in this sample.

6.2.1. THE IMPACT OF METABOLIC DISEASE ON BONE HISTOLOGY

The null hypothesis is accepted for the metabolic disease group because there are no differences in the rib histomorphometry of all skeletons that display osseous pathological changes indicative of metabolic disease and individuals without evidence of metabolic disease. However, there are differences between those with metabolic and infectious disease and only metabolic disease, so comparisons between the metabolic and infectious disease group and other disease types are included in this section for ease of discussion.

6.2.1.1. Metabolic disease and bone histology in the adult cohort

Individuals with metabolic disease show a complicated picture of bone microstructural health. The only time metabolic disease has a significant impact on bone histology is when comorbidities are at play (described later in this section). When all adults with metabolic disease are compared to all adults without evidence of metabolic disease diagnosis, there are no significant differences. The same is true when metabolic individuals are compared to those without lesions and when they are compared to those with only infectious disease. When adults with evidence of metabolic disease are compared to adults with evidence of both metabolic and infectious disease, the group with metabolic and infectious lesions shows smaller pores that are

more likely to be experiencing infilling by osteoblasts to create new osteons. This is supported by higher OPD for this group, though these differences are not significant. Additionally, no correlations were found with the presence of metabolic disease when age was controlled for in an ANCOVA.

These results are unexpected considering the recognized effect of metabolic stress on bone histomorphometry observed in other studies (Martin and Armelagos 1987; Brenton and Paine 2007). An explanation for the lack of histomorphometric differences between the combined metabolic disease group and the other groups is the overrepresentation of metabolic disease in the sample. Over half of all individuals in the adult sample show lesions indicative of metabolic disease (55.56%) while only nine are in the only infectious disease group. However, 35 individuals do not show lesions, so these comparisons can be made with more confidence. Another consideration is that individuals who have metabolic disease lesions on their skeleton and those who do not all experienced metabolic stress due to the Famine and so there is little variation in their bone microstructure because there is little variation between their metabolic states. In this case, it is those with infectious disease and those without lesions who deviate from the perceived "normal" for this population. This issue becomes more interesting when the metabolic and infectious disease group is looked at a bit more closely.

6.2.1.2. Metabolic and Infectious disease in the adult cohort

The null hypothesis was rejected for the metabolic and infectious disease group because there is a difference in the rib histomorphometry of adults that show osseous pathological changes indicative of both metabolic and infectious disease and individuals with evidence of only metabolic disease as well as those with only infectious disease. As stated earlier, bone remodeling in metabolic disease stands out only when combined with evidence of infectious disease. Recently, Roberts and Brickley described how metabolic and infectious disease can often create a feedback loop where malnutrition reduces the body's immune response leaving it more vulnerable to infection (Roberts and Brickley 2019).

However, when the group with both metabolic and infectious diseases lesions is compared with all other groups these individuals show more balanced bone remodeling. The comorbidity group

has significantly smaller porosity area and less percent porosity area than those without lesions and smaller pores than those with only metabolic disease. While other variables do not show a significant difference, individuals with metabolic and infectious disease do show more mature bone remodeling, such as OPD, than the metabolic group and the group without lesions, but this is more complicated when it is compared to the infectious group.

When the comorbidity group is compared to the group with only infectious lesions, those with comorbidities have a lower mean age, smaller osteon area, smaller porosity area, lower percent porosity area, lower percent cortical area, higher OPD, and a higher prevalence of double zonal osteons. These results lean towards less osteoclastic resorption and more mature remodeling in the group with both metabolic and infectious disease even though their mean age is slightly lower. Though none of the differences described between these two groups were statistically significant, the presence of scurvy within the metabolic disease group may have affected the bone remodeling more than the presence of infectious disease. Since the prevalence of scurvy in the comorbidity group is high (83.3%) and scurvy is associated with less porosity and more mature remodeling in this group (discussed in the next section), if Vitamin C has been reintroduced into the diets of those with evidence of scurvy, osteoblasts would be able to resume collagen deposition and activate bone remodeling that appears to be more balanced than those with infectious disease and those without lesions.

6.2.1.3. Scurvy and bone histology in the adult cohort

Overall, those with scurvy showed smaller osteon area, lower percent cortical area, higher Haversian canal circularity, and lesser porosity area and percent porosity area than those without scurvy, though the last two variables were not statistically significant. These results indicate more advanced remodeling and less porosity in those without scurvy. However, the test for differences between adults showed significantly less cortical bone for those with scurvy in each group (only metabolic, metabolic and infectious, and all metabolic combined). Reduced cortical bone in individuals with scurvy lesions is consistent with studies that link scurvy to cortical thinning as observed through radiographs (Resnick 1995; Fain 2005). It is likely that cortical thinning occurs because Vitamin C deficiency reduces osteoid deposition, a necessary component for bone building. If osteoclast resorption continues but osteoid deposition is

inhibited, then the cortical bone will become thin and porous. This was the case in a study examining the histology of scurvy induced guinea pigs where Kipp and colleagues recorded lower bone volume and a higher number of osteoclasts on the bone surface of animals with diets low in Vitamin C indicating that bone maintenance may be disrupted, and mature bone may form at a slower rate in individuals with scurvy (Kipp et al. 1996). However, the Kilkenny Union Workhouse sample shows more mature bone in those with scurvy.

Additionally, while the increase in osteoclast resorption observed in other studies indicates scorbutic individuals will have greater porosity than non-scorbutic individuals, in this study, scorbutic individuals with only metabolic disease and when groups are combined have smaller pores and less porosity than adults without evidence of scurvy. These results indicate this group was not as affected by hyperactivity in osteoclastic resorption. However, since the development of scorbutic lesions depends on collagen deposition for bone formation, it is thought that Vitamin C must be integrated back into the diet for layers of new bone to form (Brickley et al. 2020). It is possible that the reintroduction of Vitamin C that allowed for lesions to form also allowed for the deposition of osteoid that mineralized into cortical bone and began to fill in existing pores within the cortex, reducing their size and the amount of porosity. If this happens quickly, it may explain why there is more dense bone in those with evidence of scurvy and may explain why there are slightly more double zonal osteons in those with scurvy. Though this is not significant data, it is inconsistent with reports of increased bone resorption and risk of osteopenia in individuals with lesions indicative of scurvy (Resnick 1995; Fain 2005).

These results showed that the presence of scurvy is associated with lower bone porosity; however, the metabolic and infectious disease group is different. In the metabolic and infectious group, those with macroscopic lesions indicative of scurvy show larger pores and more porosity than those without. All individuals in this group suffered from metabolic and infectious disease but out of a total of twelve individuals with scurvy, eight also had sinusitis, and three had rib lesions which are not specifically diagnostic of any disease but may indicate a respiratory illness. Respiratory illnesses are known to affect the porosity of ribs and the shape of the ribs in the region of the infection (Roberts and Brickley 2018) and may have increased the prevalence of porosity for those with scurvy in the metabolic and infectious disease group.

Additionally, while only one adult showed evidence of adult rickets or osteomalacia and just two were recorded as having osteopenia, nutritional deficiencies active at the time of death that also inhibit the function of collagen mineralization such as Vitamin D deficiency, calcium deficiency, or pellagra (discussed later) may have cause porosity by affecting the mineralization of bone collagen. For example, a study showing the effects of Vitamin C supplements on the bone mineral density of women with calcium deficiency found that supplements were not effective in increasing bone density without adequate calcium intake (Hall and Greendale 1998).

Within the only metabolic group, the scorbutic adults also had smaller and more circular osteons. In the metabolic and infectious disease group, those with scurvy showed the same trend with greater OPD, which indicates more mature remodeling than the group without scurvy, despite the increased porosity in the scurvy group. These trends contrast with the study by Kipp and colleagues that showed slowed bone remodeling in scorbutic bone histology, though the reduction in %Ct.Ar for both of these groups agrees with the study and may imply a prior upset in the coupling of bone formation and resorption (Kipp et al. 1996).

6.2.1.4. Metabolic disease and bone histology in the subadult cohort

Subadults that only show evidence of metabolic disease had significantly lower percent cortical area and lower OPD than those with evidence of metabolic and infectious comorbidities. Children with metabolic and infectious disease are older (mean age =12.14 ±2.54 years, n=62) than those with only metabolic disease (mean age =8.01 ±4.76 years, n=7), so these results are expected since they are consistent with age associated increases in cortical bone and OPD over time. In children, larger pores and higher percent porosity were also observed for the group without metabolic disease compared to those with metabolic disease. These results were not significant, but they do show evidence for less osteoclastic resorption in the younger group suffering from only metabolic disease. This is unusual as porosity is often observed in the youngest children in previous studies and decreases with young adult age before increasing again in old age (Pfeiffer 2006; Streeter 2010).

6.2.1.5. Metabolic and Infectious disease in the subadult cohort

The group with comorbidities exhibited significantly greater percent cortical area and higher OPD than those with only metabolic disease. As discussed above, this group also had significantly lower porosity area as well as lower percent porosity area than the individuals without lesions, though the latter is not significant. These results are expected since porosity fills in with increasing age in children and in this group, individuals with metabolic and infectious disease ($\bar{x}$ =12.1 ±2.54 years, n=7) are older than those without lesions ($\bar{x}$ =10 ±3.69 years, n=15). This pattern was also observed when the metabolic and infectious disease group was compared to those with only metabolic disease, but this observation was not statistically significant. The comorbidity group is older than both the group without lesions and the metabolic group by about two and three years, respectively, and appears to be maintaining patterns of bone turnover as expected for the differences in age. While it appears this group is following a similar patterns to the adult cohort, variation in subadult bone includes increased porosity for the youngest children and may not be a result of disease factors.

6.2.1.6. Scurvy and bone histology in the subadult cohort

In the subadult cohort, those with only metabolic disease who show signs of scurvy demonstrate smaller osteon area than those without scurvy. This is the only significant difference between the two groups; however, those with scurvy show less porosity area, lower percent porosity, and lower percent cortical area. This is unexpected due to the difference in age between those with scurvy ($\bar{x}$ =7.6 ±4.66 years, n=58) and those without scurvy ($\bar{x}$ =13.38 ±2.84 years, n=4) in the metabolic group and the expectation that younger children will have greater porosity. These trends are consistent with those observed in the adults and may indicate those without scurvy are experiencing greater metabolic stress than those with scurvy. Subadults who exhibited lesions indicative of scurvy also had lower OPD, but this may be because there is a higher frequency of young children in the scurvy group than in the group with both infectious and metabolic disease.

The presence of scurvy throughout the subadult cohort may indicate generalized resilience throughout the population, especially in cases where infectious disease is involved. This is supported by the histological evidence that showed the most balanced bone microstructure (less

porosity, greater cortical area, and greater OPD) for subadults with both metabolic and infectious disease where all seven children showed evidence of scurvy. If these children were recovering from scurvy after being provided with relief food in the workhouse, this may explain why this group appears to have healthier bone microstructure, albeit with less cortical bone than expected, and why they were able to survive long enough to form infectious lesions. Geber observed the lowest frequency of lesions in the youngest children in the sample (neonates and infant skeletons) and the highest rate of lesions in the young children and adolescents (Geber 2015). In this sample, those with scurvy showed surprisingly healthier bone microstructure than those without scurvy which may provide support for Geber's interpretation of resilience in those who lived long enough to produce lesions indicative of scurvy.

The following case study showed the effect scurvy had on a young male who was excavated from the Kilkenny Union Workhouse grounds. The case includes personal details such as clay-pipe facets and histological evidence of continuous metabolic disruption, features that provide insight into the multifaceted human lives that were lost during the Great Famine.

6.2.1.7. Case Study: Burial CCCVIII

Individual CCCVIII is a young adult male with a point age estimate of 22.8 years old (Figure 6.1). At the time of analysis, the skeleton was in excellent condition and 98% complete and stature was estimated at 164cm (5ft 4.6in). Dental analysis revealed this individual had clay-pipe facets on their maxillary and mandibular lateral incisors and canines. These facets indicate prolonged pipe smoking, but the dentition was in good shape otherwise, with only slight calculus and slight dental attrition present. This person had lesions indicative of scurvy with porosity on the ectocranial surface of the left greater wing of the sphenoid. The rib bone histology shows this person had significantly larger porosity area and greater percent porosity within their cortex than the adult population means. This is consistent with other adult scurvy data which shows significantly more porosity for those with Vitamin C deficiency than those without scurvy. It is also consistent with the age data which shows young adult males with more porous bones than their older counterparts. There is also a very high frequency of double zonal osteons in this person's rib, which indicates repetitive disruption of bone mineralization (Figure 6.2).

Figure 6.1. Burial CCCVIII excavated on the grounds of the Kilkenny Union Workhouse. Photo by Margaret Gowen and Co. Ltd.

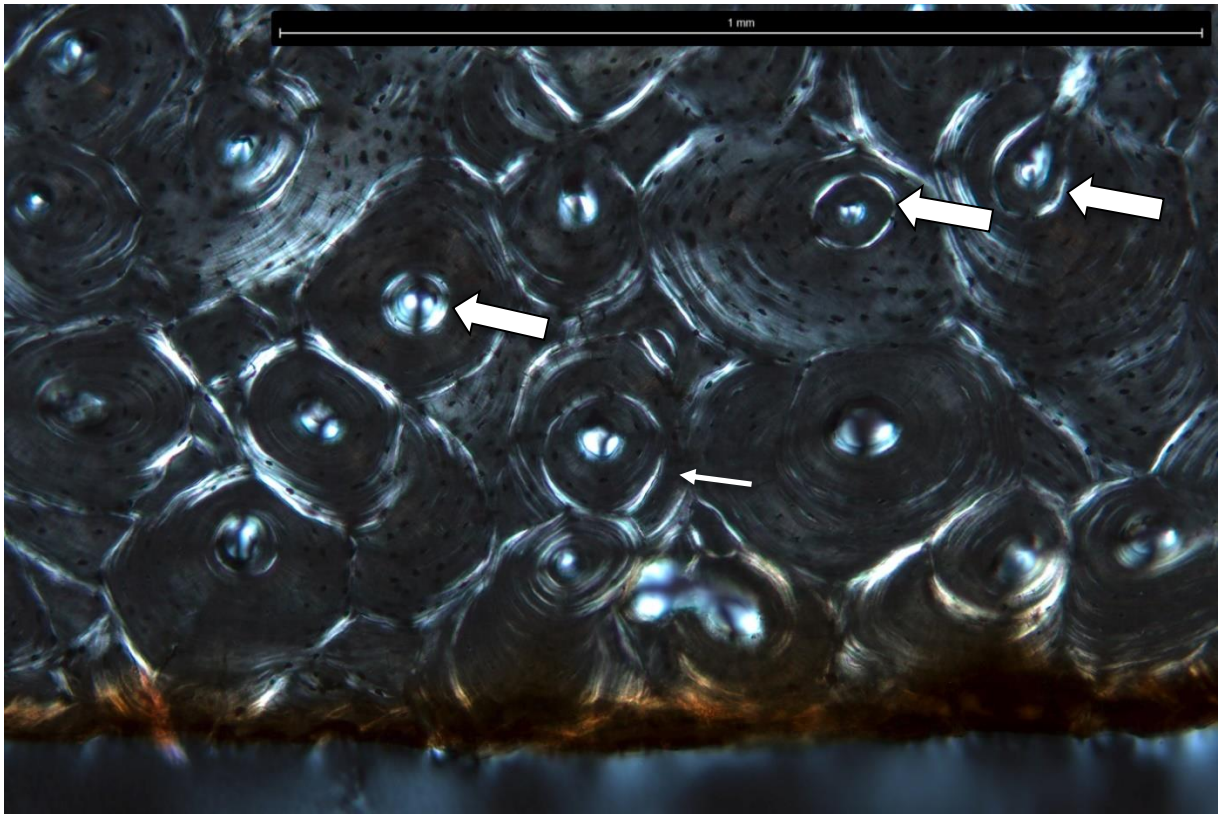

Figure 6.2. The middle rib of CCCVIII showing multiple double zonal osteons (thick white arrows) and a Type II osteon (thin white arrow) (10x magnification under polarized light; scale set at 1 mm)

This person also exhibits Schmorl's nodes and slight ossifications of ligamenta flava (OLF) on their thoracic vertebrae, which may reflect physical activity that gives a certain insight into his life experience. Schmorl's nodes are common spinal pathologies often associated with older age, weakening of the spine due to pathological conditions, and a history of physical labor

(Kyere et al. 2012). While OLFs are understudied, they are also associated with age and coincide with the presence of Schmorl's nodes, indicating a potential association with activity as well (Geber and Hammer 2018). In the Kilkenny Union Workhouse sample, OLFs were observed in 83.6% of individuals and most were reported in adults over the age of 25. There were no sex differences except in the 36-45-year-old age group where men were more likely to have OLFs than women.

Public works ended in April 1847, just four months before the first people were buried on the grounds of the Kilkenny Union Workhouse. When public works were beginning to shut down in March, the workhouses became inundated with people seeking indoor relief. In May, soup kitchens were established so that outdoor relief could continue to be provided. As mentioned in Section 3.3.1, these kitchens distributed a meal sufficient to slow the rate of deaths (Crawford 1984) but lasted a brief six months and the soup kitchen relief scheme ended in September when the workhouses would again see an increase in the inmate population (Grey 2012).

As a young male, individual CCCVIII would only be accepted into the workhouse under dire circumstances but it is possible this person was suffering from Vitamin C deficiency and other forms of malnutrition while participating in public works. After the closure of the works, they may have been supplemented with nutrients from the stir-about, but this would not have provided a sufficient amount of Vitamin C required to cure scurvy. Once the soup kitchens were shut down, there were few options besides entering the workhouse. However, at some point, a meager vegetable soup was distributed within the institution (Geber et al. 2019). This soup usually consisted of rice, oatmeal, turnips, parsnips, onions, salt, and pepper (Board of Guardians 1848). Those who were suffering from scurvy may have obtained some relief due to the ascorbic acid content of turnip greens and tops, which contain 62mg/100g and 46mg/100g, respectively, when fresh but retain only 36% of ascorbic acid when steamed and lose all Vitamin C content when boiled (Francisco et al. 2010). Parsnips, which can contain between 15-30mg of Vitamin C per 100g and tend to maintain their nutrient content when boiled and steamed (up to 91% and 86%, respectively) (Brown and Fenton 1942) may better explain the presence of scorbutic lesions indicating some form of relief was introduced to stimulate bone

remodeling, but would also explain the presence of porosity from a lack of collagen deposition due to malnutrition since the scurvy would have proliferated when the soup was not available.

It is not possible to know if this person received maize as a relief food because their isotope data has not been analyzed, but it is highly likely due to what is known about the distribution of relief food in the workhouse. The potential for pellagra to be present in this population and the possibility that some skeletons diagnosed as having scurvy suffered from pellagra is likely due to the similarities in the manifestation of cranial lesions in both disease types and the similarities in the effect on bone remodeling for both diseases (Brenton and Paine 2007). This is described in more detail in Section 6.3.3.

6.2.2. THE IMPACT OF INFECTIOUS DISEASE ON BONE HISTOLOGY

The null hypothesis was rejected because there is a difference in the rib histomorphometry of skeletons that display osseous pathological changes indicative of infectious disease and individuals without evidence of infectious disease.

6.2.2.1. Infectious disease and bone histology in the adult cohort

This research shows that adults with skeletal evidence of infectious disease have more balanced bone remodeling than those without infectious lesions. This is the case when those with evidence of infectious disease (including those with comorbidities of infectious and metabolic disease) are compared to those without infectious disease. For example, porosity area, a variable that is used to infer the quality of bone health, was significantly lower in individuals who showed evidence of infectious disease than in those without infectious lesions. Porosity is widely considered to be associated with increased age; however, porosity did not correlate with age in this group. This was unexpected due to the observed correlation between porosity and age in other groups but may indicate disease is affecting this variable by limiting the capacity of the rib to maintain a balance in remodeling.

Even though there was not a correlation with age for porosity in the adult cohort, the infectious disease group showed a stronger correlation with age for both porosity area and percent porosity than the group without lesions. This is inconsistent with a study by Ruchonnet who found that

pathological individuals showed a weaker correlation with age than non-pathological individuals (Ruchonnet 2019); however, it is known that all individuals in this group suffered from some form of Famine induced stress and the lack of lesions does not necessary mean there is a lack of disease. In fact, this study has shown, through analysis of porosity across multiple disease groups, that bone turnover was better maintained in those that show evidence of disease than those that do not. Individuals who exhibit infectious lesions also have histomorphometric means that indicate more mature bone remodeling when compared to those without evidence of infection. While these variables were not significantly different, when compared to those without lesions, individuals with infectious disease also showed smaller and more circular osteons and Haversian canals, lower percent porosity, greater percent cortical area, lower frequencies of double zonal osteons, and higher OPD which is indicative of greater remodeling when there are no significant differences in age between groups. Those with infectious disease also show most of these patterns when they are compared to those with only metabolic disease suggesting that osteoid deposition and mineralization was better maintained in those with evidence of infection.

It is believed that most deaths that occurred during the Great Famine were due to infection rather than starvation alone, particularly those who died in the Workhouses or the associated infirmary (Ó Gráda 2012). If infectious disease was the primary reason people died, then their ability to fight infection was dependent on their innate and adaptive immune responses as well as the presence of other comorbidities (Roberts and Brickley 2018). While the appearance of less porous bone in those with infectious disease may signify a healthier individual, it is possible the absence of porosity indicates less time between the onset of disease and death, such as in the case of acute infectious disease. Since the skeleton acts as a reservoir for the nutrients the body depends on for survival, the lack of bone density in those without lesions may indicate a longer period between the onset of disease and death where the body was retrieving nutrients from the skeleton, creating more porosity over time. As discussed earlier, this may be a function of the Poor Law system wherein “healthy” able-bodied males and females were not granted access to the workhouse and instead allocated public relief works building roads and drainage systems for small wages, which would have resulted in more energy expenditure with little nourishment to replenish their bodies. Around the time the workhouse grounds began to be used

for intramural burials, the public works shut down causing many younger men and women to enter the workhouse, exposing their already weakened bodies to greater risk of infection.

6.2.2.2. Infectious disease and bone histology in the subadult cohort

The results of this research found that individuals with infectious disease have slight trouble maintaining bone remodeling. Children with lesions indicative of infectious disease (only infectious group and metabolic and infectious group) have significantly lower percent porosity area, which is expected due to their older age ($\bar{x}$ =12.70 ± 2.49 years, n=10) relative to all those without infectious lesions ($\bar{x}$ =8.39 ± 4.61 years, n=77) and the greatest percent cortical area. However, the three individuals in the group with only infectious disease have a mean age of 14 years, the highest mean in of all the disease groups, but this group also has the lowest OPD and the largest osteons of all disease types, which is not expected since OPD increases with age while osteon size is known to decrease with age. These results indicate low porosity but reduced remodeling activity in subadults with infectious disease.

The results obtained for individuals with infectious disease in the subadult cohort are in line with the results obtained in the adult cohort where adults with infectious disease appeared to have the least porous bone. However, these adults also show the most mature bone remodeling, which is contrary to the more immature remodeling present in the subadults for this sample. During the Famine, destitute children were allowed in the workhouses but those with parents were often turned away. Anecdotes from the period include reports of children purposefully orphaned by their parents who hoped their children would be better taken care of in the workhouses, which may explain why they were able to retain bone better than those who would have lived outside the workhouse. Children with infectious disease are the oldest of the sample, which may be interpreted as a group with more resilience, a hypothesis that is supported by the results discussed in the next section regarding poor bone maintenance observed in those without lesions who are also the youngest of the group.

6.2.4. THE IMPACT OF LESION PRESENCE ON BONE HISTOLOGY

The null hypothesis was rejected for the group without lesions because differences were found in the rib histomorphometry of skeletons that do not have osseous pathological changes indicative of disease and other disease groups.

6.2.4.1. Bone histology and the absence of lesions in the adult cohort

The rib bone microstructure of adults without lesions has the most porotic and immature bone of the sample. When compared to the metabolic and infectious disease group, those without lesions had significantly more porosity and greater percent porosity area. While not significant, this group also had more double zonal osteons and lower OPD when compared to all other disease categories. Those without lesions have the second highest porosity area behind the metabolic group but also had the second highest percent cortical area behind those with only infectious disease. The amount of cortical area in this group may be related to the lack of bone maturity or it could be related to a low level of osteoclastic resorption, though the amount of porosity relative to other disease groups suggests high levels of active resorption at the time of death. The high frequency of double zonal osteons is interesting because it implies multiple instances of disrupted mineralization throughout the last few years of life (Martin and Armelagos 1985). The recent appearance of double zonal osteons that have yet to be remodeled may be a product of a recovery from nutritional deficiency in the years before death, perhaps due to workhouse provisions. This data and the relative histological maturity of this group may also be a response to seasonal famines over the lifetime where those without lesions may represent a resilient group who have spent many years battling metabolic stress before entering the workhouse. Alternatively, the lack of lesions in this group may imply a swifter death for a vulnerable group of people. If this is the case, it is unlikely they suffered from scurvy as the presence of double zonal osteons would mean collagen deposition was occurring before death. However, there is more to learn about double zonal osteons and if remodeling is not occurring due to Vitamin C deficiency, then it is possible these osteons formed before scurvy set in.

6.2.4.2. Bone histology and the absence of lesions in the subadult cohort

In the subadult cohort, individuals without lesions have more porosity than any other disease group; however, this difference is only statistically significant when compared to the infectious

and metabolic disease group. While not significant, this group has the largest pores of all disease types excluding the only infectious disease group. Unlike the adult cohort, this group does not appear to have more immature bone remodeling than the other disease types and, except for porosity, there are no real differences between those without lesions and the other disease types. As this group is the second youngest of the disease types, the presentation of poor bone health in this group may indicate increased vulnerability for children without lesions.

6.2.5. QUESTION 1 SUMMARY: INTERPRETING WORKHOUSE POPULATION FRAILTY USING BONE HISTOLOGY

At the time of its opening in 1842, many of the people who entered the Kilkenny Union Workhouse would have already been suffering from nutritional deficiencies due to poverty and homelessness (Crawford 1984; Clarkson and Crawford 2001). In fact, as discussed in Chapter 3, it was a requirement that those who enter the workhouses be destitute and without alternative options for relief (Irish Poor Law Act 1838). When the blight arrived and the Great Famine began, the workhouses started to fill with people who had recently become destitute and were in need of relief after the failure of the potato. In April 1847, the Russell administration in Parliament ended the public works system and amended the Poor Law in June to include the Gregory Clause (Smyth 2012). This change caused many more people to become destitute and enter the workhouses after they were made homeless by evictions, pushing the population of the institutions well past their capacity, limiting the available relief food supply, and increasing the risk of death due to disease (Smyth 2012). Factors such as the changing government policies regarding famine relief, the age and sex of the poor, and the conditions present within the workhouse all played a role in the variations of disease observed between individuals in this sample.

As stated in Section 1.2., one of the goals of this thesis is to determine if bone histomorphometry could help tease out the meaning of lesion variation by comparing disease types with remodeling patterns in the rib. Since rib bone histology provides a retrospective picture of bone health, the presence of statistically significant differences in histomorphometry between people with various disease experiences provided information about their health status prior to their entry into the workhouse.

Often, skeletons with lesions indicative of disease are understood to be the most vulnerable in the population; however, identification of disease in skeletal populations can be limited by early death. For example, if a person dies from a sickness that may have caused skeletal lesions if it had time to progress. This leads to a complex issue in paleopathology, which is the interpretation of the osseous lesions. The question of whether lesions observed in bone are indicative of highly resilient individuals who survived long enough to develop lesions or if lesions are indicative of frail individuals is known as the "osteological paradox" and has been debated for nearly three decades as discussed in Section 2.5.4.2.2. (DeWitte and Stojanowski 2015; Wood et al. 1992).

While the effect of the osteological paradox on the interpretation of health in archaeological assemblages is dependent on the research questions at hand, greater clarity in the role and expression of genetic and biological predisposition to disease is warranted (DeWitte and Stojanowski 2015). Frequencies of infectious disease in pathological collections are often higher in the historical records than the frequency of disease that is recorded from osteological collections (Roberts and Brickley 2018). Brickley and colleagues comment that the high mortality rate of individuals living in overcrowded spaces during episodes of food shortage are due to acute infections that result from lower immune response. As discussed in Chapter 2, these diseases often do not show on the macroscopic surface of the bone (Roberts and Brickley 2018; Brickley, Ives, and Mays 2020)). As a result, actual experiences of infectious disease are not recorded in the paleopathological analysis.

This was the case in the Kilkenny Union Workhouse where skeletal evidence of infectious disease was low (11.1%) but the number of individuals without evidence of lesions was high (88.9%) (Geber 2015). One way to explore this issue is through observation of the histological response of bone to disease. Even if pathological lesions do not form, many diseases affect bone formation and remodeling processes throughout the skeleton, which may be observable through bone histological analysis (Albright and Reifenstein 1948; Brickley, Ives, and Mays 2020) as observed by Robbins Schug and Goldman (2014) in the study of young children with shorter

stature and fewer lesions from the Late Jowre phase of Prehistoric India (1000-700 BC) who did not maintain or acquire the bone mass expected for their age.

While starvation itself was a major contributor to metabolic disease for the most destitute in mid-nineteenth century Ireland, infectious disease knew no class. Many clergy, medical, and "middle class" people who either served in the workhouses or entered them as a consequence of the economic decline, died because of infection. Entry into the Kilkenny Workhouse meant receiving rations of food and a roof over one's head, but it also meant entering a crowded institution rampant with diseases. If a young man entered the workhouse after receiving regular outdoor relief food or purchasing his own food after working in the public works system and was subsequently infected, it is possible that he would not have any evidence of disease on his skeleton. This same individual may have suffered from and showed evidence of metabolic disease but not lived long enough with infectious disease for it to cause lesions. More comfortable tenant farmers who would have had access to nutritious foods throughout their lives may have been metabolically stable but succumbed to infectious disease upon entering the workhouse when the potato crop continued to fail.

The Kilkenny Union Workhouse opened seven years prior to the start of the Famine and nine years prior to the first use of mass burials on the grounds. Adults and children with infectious disease but without other indicators of disease may represent a group of people who were admitted into the workhouse before crowding became an issue. Often, people who were admitted into the workhouse infirmary before the famine ate well. They were provided with potatoes, bread, and milk as treatment for their ailments which would have satisfied many of the nutritional demands needed to improve their health and maintain proper bone turnover. However, if these individuals were suffering from tuberculosis, for example, a decline in the availability of food, lack of continued care, or another infection due to overcrowding may cause a decline in health that led to their death.

Overall, the adults and children of the metabolic and infectious disease types were more adept at maintaining bone than the other disease groups for both cohorts. However, while adults with infectious disease appeared to have healthy bone maintenance, this group had the most

immature bone in the subadult cohort, despite having the highest mean age. Additionally, those with scurvy were better at maintaining bone and showed evidence of more regular patterns of bone remodeling than those without scurvy. It has been hypothesized that the appearance of healthy bone remodeling in the scurvy group compared to other disease groups may be a function of recovery due to the reintroduction of Vitamin C in the diet from workhouse relief but historical records state that relief food during the famine mostly consisted of oatmeal, milk, and maize consumed in its various forms (Crawford 1984). However, a record of soup with some vegetables was recorded in the minute books by the Board of Guardians. This was confirmed by analysis of dental calculus of teeth from the Kilkenny population performed by Geber and colleagues (Geber et al. 2019). This evidence supports the idea that these individuals were likely receiving some relief food that contained the little amount of Vitamin C necessary to reignite collagen synthesis.

It is possible that those who suffered from scurvy also suffered from the Vitamin B3 deficiency disease pellagra. As described in Chapter 3, pellagra often exists in populations that subsist on maize-based diets. However, skeletal manifestations of pellagra are understudied, and it is not possible to know whether the pathological changes indicative of scurvy are also pathognomonic for pellagra. The next section discusses the correlations in histomorphometry between the values of $\delta^{15}N$ isotopes, which may be indicative of starvation, and levels of $\delta^{13}C$ isotopes, which may be indicative of maize consumption and serves as a proxy for pellagra. It also describes evidence that the inmates who were buried on the grounds of the Kilkenny Union Workhouse suffered from pellagra.

Generally, it seems that histological patterns in adult and subadult ribs align with the osteological paradox, where adults and children without lesions are the most vulnerable. The strongest evidence exists where poor bone maintenance was observed in the young children without lesions and less porosity existed in the group with metabolic and infectious lesions. Also, the presence of low porosity with more mature remodeling in adults and subadults with lesions indicative of scurvy and the high frequency of double zonal osteons in adults with scurvy provide support for experiences of recovery from metabolic stress. However, if pellagra was misdiagnosed as scurvy, the picture becomes more complicated.

6.3. ADDRESSING QUESTION 2: HISTOMORPHOMETRIC COMPARISONS OF DIETARY STABLE ISOTOPES

Correlations between dietary stable isotopes and histomorphometric data exist between $\delta^{15}N$ and osteon size in adults and between C_4 values and the size of Haversian canals in subadults. Stable isotopes are a reflection of food and water consumption during the formation of tissues and can inform dietary patterns over time depending on the tissue being analyzed (DeNiro and Epstein 1981). Isotopes have also been useful in bioarchaeology for revealing periods of stress in adults and children from the past. For example, geographic relocations and other stresses such as drought, flooding, or famine that cause food scarcity can be observed in elevated levels of nitrogen; and some major transitions in diet due to events like the move to agriculture or the consumption of imported relief food may be reflected in the level of carbon values (Beaumont and Montgomery 2016; Miller et al. 2020a; Redfern et al. 2019; Vogel and van der Merwe 1977). When examining isotope ratios to determine diet from the bone collagen of adults, Beaumont and colleagues emphasized the importance of considering age-related changes in bone remodeling and the relatively slow rate of bone turnover in adults compared to children (Beaumont et al. 2013). The results of this study show that remodeling in adult ribs was affected by nutrient deprivation, while subadult ribs were impacted by the consumption of relief food.

6.3.1. STABLE ISOTOPE CORRELATIONS WITH BONE HISTOLOGY IN THE ADULT COHORT

A significant positive correlation was observed between $\delta^{15}N$ and osteon area. This correlation was expected based on prior research that observed reduced remodeling in individuals with evidence of nutritional deficiency, including pellagra, when age estimation methods were applied (Paine and Brenton 2006). Though it is surprising that more variables did not correlate with the rise in $\delta^{15}N$, this increase was slight and non-significant when compared between individuals within the sample (Beaumont et al. 2013). The bulk rib collagen sample analyzed by Beaumont and colleagues showed the Kilkenny Union Workhouse sample as having the lowest average $\delta^{15}N$ values than other comparative nineteenth century sites (Beaumont et al. 2013). Beaumont and colleagues speculated that the slow turnover rate of bone relative to the onset of the Famine and the time of death thereafter is responsible for the observation of low $\delta^{15}N$ values when evidence of starvation (as represented by elevated $\delta^{15}N$ values and contextual

information about diet) was expected. Metabolic disease is present in 59% percent of the sample used to answer this question and nearly 30% exhibiting no lesions. If metabolic stress inhibits bone formation as the research in the present study and other studies have shown, then Beaumont and colleagues' speculations are supported, and it will take longer for new collagen to represent dietary changes in individuals with pathological lesions indicative of metabolic disease (Beaumont et al. 2013). However, when incremental dentine from childhood was analyzed for these adults, $\delta^{15}N$ levels co-varied with $\delta^{13}C$ indicating a small change in diet during childhood. This may be indicative of seasonal famine, but it could also indicate the consumption of protein, which contradicts much of the historical understanding regarding the diet of the poor in Ireland, but is supported by the research by Geber and colleagues who found some protein in the dental calculus of individuals from the Kilkenny Workhouse (Geber et al. 2019). When examined by Beaumont and colleagues, adult rib bone collagen also recorded a slight trend toward the shift from C_3 potatoes to C_4 maize but the histomorphometric values did not replicate this trend (Beaumont et al. 2013). This is likely due to the turnover rate as described above, but it is interesting that the changes in isotope values would be reflected in the collagen without a corresponding trend in histological variables, especially since there was a trend in osteon size for $\delta^{15}N$.

6.3.2. STABLE ISOTOPE CORRELATIONS WITH BONE HISTOLOGY IN THE SUBADULT COHORT

This study found no correlations between $\delta^{15}N$ values and histomorphometry of the ribs in the subadult cohort, but there was a significant positive correlation between $\delta^{13}C$ values and Haversian canal area. This data suggests Haversian canal size may be impacted by the consumption of maize. As reported in a previous study by Beaumont and colleagues (2013), bulk rib bone collagen showed evidence of C_4 in the children buried on the workhouse grounds. In the present study, Haversian canal area increased with the rise in $\delta^{13}C$ values towards the C_4 range. However, there is much variation present in the data and $\delta^{13}C$ values may not contribute much to the variation observed in Haversian canal area. It is likely that this correlation a result of chance and impacted by the small sample size; regardless, it is important to consider the significance of this finding.

While the size of Haversian canals has been incorporated into studies of histomorphometry regarding the response to biomechanical forces and variation across age (Botha, Lynnerup, and Steyn 2020; Burr, Ruff, and Thompson 1990; Epker and Frost 1965; Schlecht et al. 2012; Skedros et al. 2013), little attention has been given to their variation with metabolic changes (Paine and Brenton 2006). Haversian canals house the vessels that transport blood and the sensory communication network throughout every bone in the body (Jaffe 1929) and can be influenced by diseases that affect the blood vessels such as atherosclerosis (Wolff 1950). Reichert and Mulhern noted that Haversian canals were smaller in double zonal osteons and suggested this may have to do with an adaptation to the restriction of resources (Reichert and Mulhern 2018). On the other hand, the study by Paine and Brenton showed higher Haversian canal area in individuals that were known to suffer from pellagra, which is consistent with the data found in this study (Paine and Brenton 2006). Additionally, Those with C4 values in this group show lesions consistent with those present in groups known to have suffered from pellagra including cribra orbitalia (CDXLIV), fine porosity on the endocranial surface of the greater wings of the sphenoid and the endocranial surface of the squamous portion of the temporal bones, and extensive alveolar porosity on both the maxilla and mandible (Miller et al 2020; Paine and Brenton 2006).

Out of the eight individuals with Haversian canal area data in this subsample, all but two show evidence of scurvy. If the hypothesis by Reichert and Mulhern is considered, this increase in Haversian canal size in children with higher $\delta^{13}C$ values may be an indication of better health in subadults when relief food was provided (Reichert and Mulhern 2018). Alternatively, since scurvy is associated with subperiosteal hemorrhaging, the relationship between blood vessel development and Haversian canal size in scorbutic individuals should be further explored. Haversian canal size and shape is often associated with osteon size and shape, but when remodeling is not occurring (observed by smooth edges in the canal) the association between Haversian canal size and metabolic changes should be considered. Careful consideration should also be given to the data collection of Haversian canals so that remodeling events (resorption bays) are distinguished from Haversian canals and do not influence the data. For example, Richman and colleagues looked at the relationship between resorption cavities, osteon type, and diet for three different archaeological populations with varying diets and while these resorption

cavities were described as beginning within the Haversian canal, these cavities are also representative of active remodeling sites and do not reflect resting Haversian canal size (Richman, Ortner, and Schulter-Ellis 1979).

In the subadult cohort, children without scurvy had slightly higher $\delta^{13}C$ values and slightly lower $\delta^{15}N$ values, indicating greater evidence for maize and less evidence for malnutrition, though these are not significantly different (Table 6.1). It is possible these children were receiving the minimum amount of Vitamin C necessary to prevent scurvy (10mg), potentially through a combination of nutrients from outdoor relief or soup provided in the workhouse, thus reducing their $\delta^{15}N$ value. They may also represent children who have not obtained the necessary amount of Vitamin C for lesions formation and were receiving only maize stirabout while suffering from scurvy.

Table 6.1. Values for $\delta^{13}C$ and $\delta^{15}N$ in the diet of children with and without evidence of scurvy

Scurvy		$\delta^{13}C$	$\delta^{15}N$
Not Present	Mean	-16.96	9.65
	SD	.50	1.34
	N	2	2
Present	Mean	-17.93	9.84
	SD	1.89	.40
	N	7	7
Total	Mean	-17.71	9.80
	SD	1.68	.59
	N	9	9

When subadults with and without $\delta^{13}C$ in the C_4 range are compared, children that show C_4 values have significantly larger osteon area ($t(7) = -2.77$, $p = .03$) ($M = -1.40$, $SD = .06$) than those outside the C_4 range ($M = -1.5$, $SD = .05$) and larger porosity area ($t(5) = -3.34$, $p = .02$) ($M = 1.33$, $SD = .31$) than those outside the C_4 range ($M = .75$, $SD = .15$). Those that appear to have consumed maize also have a higher percent porosity in their ribs ($M = 6.94$, $SD = 2.95$) than those that do not show evidence of maize ($M = 4.97$, $SD = .64$), though this is not significant. These data are consistent with the study by Brenton and Paine linking maize dependent diets to high cortical porosity and reduced rate of remodeling (Brenton and Paine 2007), but it is

interesting that this is the case in a sample of people who are experiencing starvation. It is possible that those who may have suffered from pellagra were in the workhouse longer than those with values outside the C_4 range. If these children entered the workhouse after the consumption of soup from the outdoor soup kitchens and subsequently began receiving maize, then they would have regressed into a diet deficient in Vitamin C, B3, and/or D and may represent the children without scurvy but with maize in their diet. However, as mentioned in Section 6.2.1.4, a soup containing turnips, parsnips, and onions may have been provided as an alternative relief food in the workhouse and if scurvy existed, this soup may be the reason scurvy lesions are present in those with more balanced bone remodeling as it would have provided a bit of Vitamin C.

The following section includes another case report that highlights some osteological features discovered on the bones of a young child with high levels of $\delta^{13}C$, indicative of C_3 values, which are consistent with potato consumption but may also be evidence of plants, grasses, or shrubs which were sometimes foraged by hungry children outside the workhouse. This may also be evidence of other relief food in the workhouse such as parsnips, turnips, or grains (Beaumont et al. 2013; Geber et al. 2019; Kohn 2010).

6.3.2.1. Case Study: Burial DXXXII

Burial DXXXII is a young child between five and six years old of indeterminate sex and stature (Figure 6.3). At the time of analysis, the skeleton was 98% complete with excellent preservation. Several skeletal pathologies were recorded during analysis, including unilateral moderate active cribra orbitalia on the left side, indicating the child likely suffered from scurvy (Figure 6.4). They also had an asymmetric fifth lumbar vertebra with alterations to the laminae including fusion with the body on the left side, which Geber identified as potentially related to trauma. This individual has the highest $\delta^{13}C$ value of all subadults in the cohort (-20.3‰) and does not fit within the C_4 range considered to represent the consumption of maize. Grasses and grains are associated with $\delta^{13}C$ values between -20‰ and -37‰ (Kohn 2010), which was likely attained due to the consumption of oats and rice in the stirabout provided in the soup kitchens or within the workhouse (Clarkson and Crawford 2001; Grey 2012). This individual also has the second highest $\delta^{15}N$ value in the cohort (10.3‰). Since the rib remodeling of children occurs

at a quicker rate than adults, it is possible this child died before receiving relief food. However, the presence of scurvy, which may cause slower bone remodeling as observed in this study, could have impacted collagen deposition and prevented the observation of maize consumption. While there is no evidence of infectious disease in this case, the child was suffering from metabolic disease due to nutritional deficiency, which would have lowered their immune response and made them more susceptible to death inside the workhouse.

Figure 6.3. Burial DXXXII excavated on the grounds of the Kilkenny Union Workhouse. Photo by Margaret Gowen and Co. Ltd.

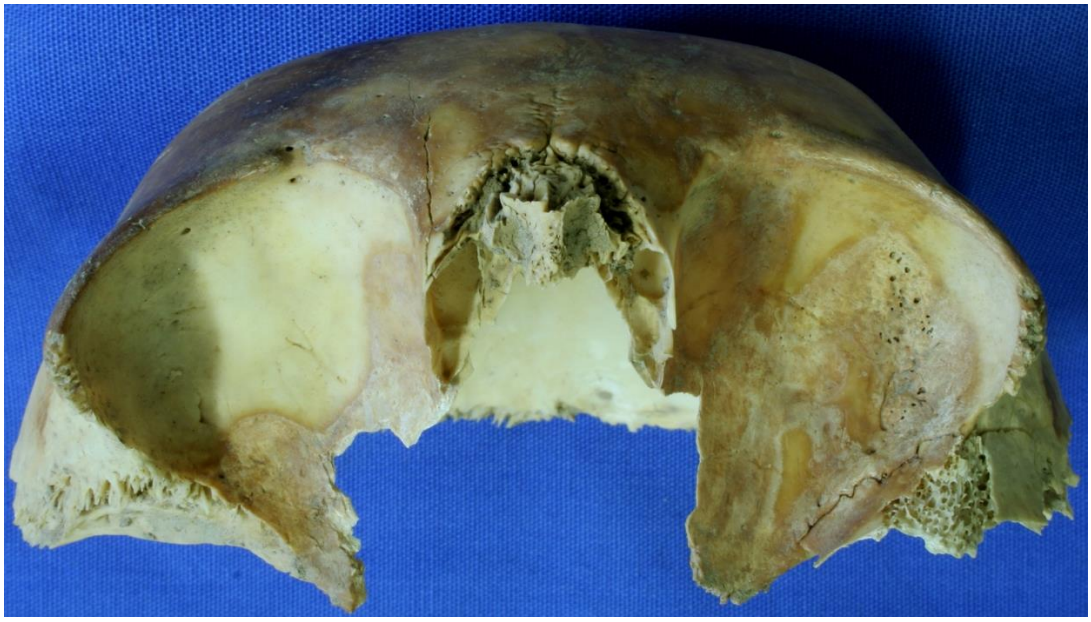

Figure 6.4. Unilateral moderate active cribra orbitalia on the left orbit of a young child from burial DXXXII. Image provided by Jonny Geber

Figure 6.5 shows a snapshot of some of the interesting features present in this young child. When compared to the subadult means for bone histology, this individual shows greater porosity area and greater percent porosity which may indicate there was an imbalance in bone remodeling. However, percent cortical area is greater in this individual than the means of the variable for the subadult cohort, indicating the size of the cortex was larger than the average child and, as previously stated, large amounts of porosity are common in young children. Additionally, this individual shows secondary osteons, a feature that is known to occur in later childhood, typically around twelve years of age (Wu et al. 1970). This was not an anomaly in this cohort and many younger children also showed secondary osteon remodeling at young ages including individuals estimated to be four years old (DXXVIII), 2.5 years old (CCXCVI), and a forming secondary osteon was even observed in a very porous 1.5 year old (CXCIII) (Figure 6.6).

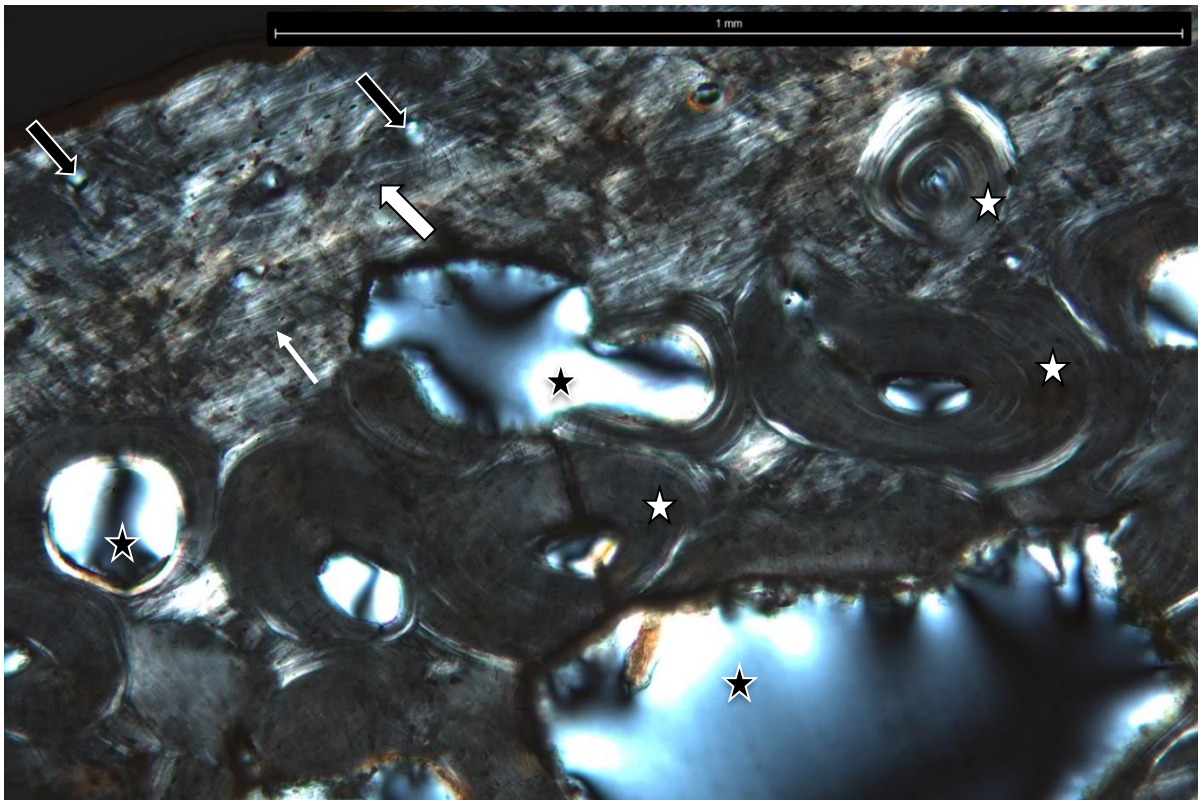

Figure 6.5. Snapshot of the cutaneous cortex of a middle rib of DXXXII showing some remaining layers of basket-like woven bone (large white arrow), sheets of unremodeled lamellae (small white arrow), primary canals (black arrows), secondary osteons (white stars), and resorption bays (black stars) (imaged under polarized light at 10x magnification; scale set at 1mm)

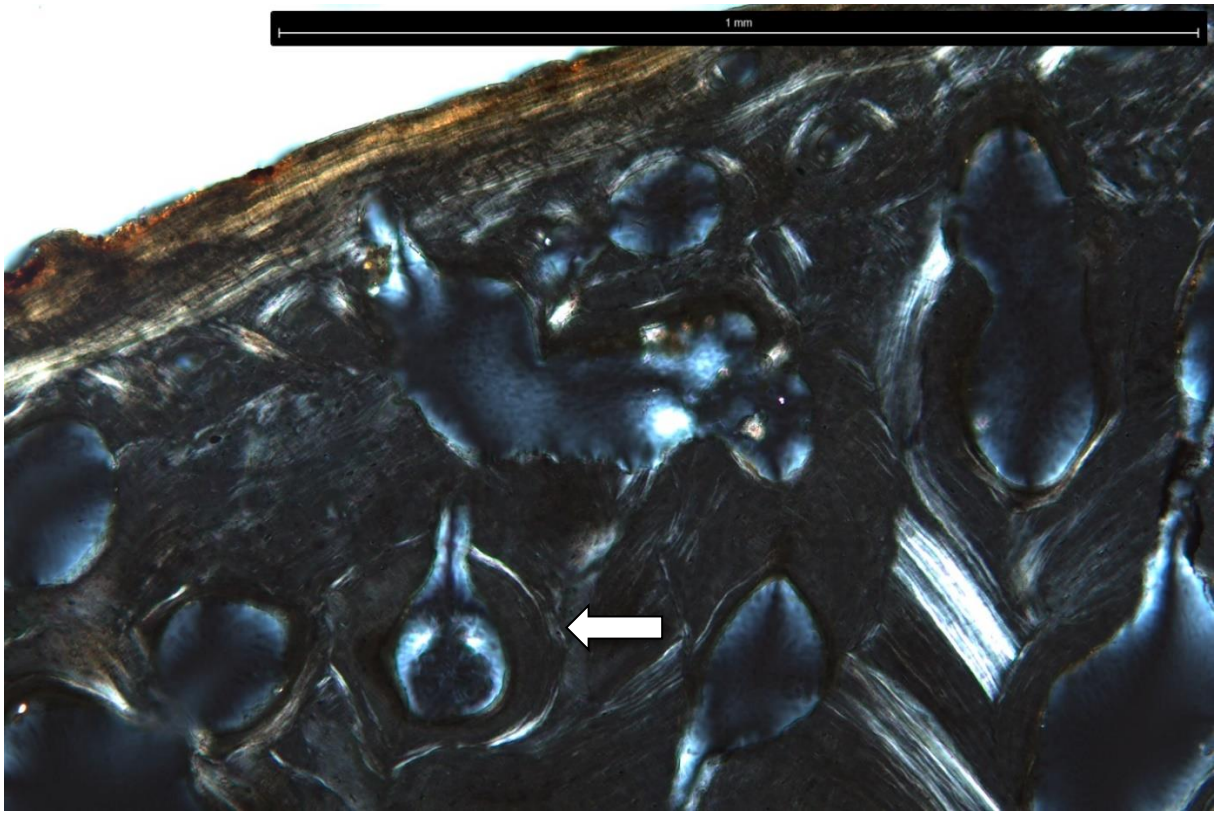

Figure 6.6. Forming secondary osteons (white arrow) in the cortex of a middle rib from a child estimated to be 1.5 years old (imaged under polarized light at 10x magnification; scale set at 1mm)

6.3.3. PELLAGRA IN THE KILKENNY UNION WORKHOUSE

As described in Chapter 3, the first D of pellagra is dermatitis. In clinical descriptions, rashes are recorded as appearing on the hands, feet, and around the neck, locations that are regularly exposed to the sun when an individual is clothed (Wan et al. 2010). However, if the person is dressed in rags or naked, as the poor were known to be (Nicholson 1851; Edwards and Williams 1956), it is possible the rash would have appeared across a greater area of the body. This may have caused more widespread cutaneous inflammation which may have been confused with other conditions such as scurvy, which causes hemorrhaging of the blood vessels and pooling of the blood under the skin (Ortner 2011). On the contrary, if the sick did not leave the bed, their skin may not have come into contact with the sun at all and rashes may not appear (Miller et al. 2020b). The Kilkenny Workhouse had inadequate ventilation and low light due to the presence of only a few small windows (O'Connor 2005) and the lack of sunlight may have prevented rashes from presenting even if the victim were not confined to their bed or the

infirmary, further complicating post hoc diagnosis based on clinical records from the workhouse.

Many of the diseases listed for the Kilkenny Union Workhouse in the 1851 Census of Ireland are specific and identifiable, but others are non-specific and recorded as generalized fever (159 males, 130 females) or diarrhea (118 males, 92 females) or other symptoms like disease of the intestines or marasmus. Death from diarrhea, which causes dehydration, could be caused by any number of diseases including cholera, typhoid, or pellagra; and insanity was a subjective diagnosis that could have resulted from any number of comorbidities of nutritional deficiencies. However, those who experienced diarrhea and dementia (or "insanity") may have been victims of the second and third "D" in the list of symptoms of pellagra (Miller et al. 2020b).

W.P. MacArthur has studied the accounts by Dr. Reid, a physician at the Cork Street Hospital in Dublin who diagnosed 970 individuals with typhoid due to the presence of fever, a "brown and dry" tongue, and neurological symptoms in which the brain was described as "a good deal involved" (MacArthur 1956). However, MacArthur argues the severity of the disease (10% mortality) and the duration of the disease (15-20 days) is not characteristic of an "attack of typhoid". Instead, the author suggests the patients may have experienced typhus without a rash or relapsing fever without the relapse. Although Dr. Reid never mentioned diarrhea in the patients diagnosed as suffering from typhoid, the two symptoms he does mention (brown tongue and mental illness) are symptoms of pellagra.

The presence of a dry, dark tongue is noted in early literature concerning the synthesis of niacin (originally known as nicotinic acid) for recovery in pellagra patients (Jukes 1989). In the 1930s, Henry Sebrell and colleagues determined that dogs who developed a "black tongue" after induced pellagra could be cured by the vitamin nicotinic (Sebrell 1981). Coincidently, another group of scientists led by Douglas Frost were working to identify a cure for the "black tongue disease" they had also experimentally induced in a group of dogs (Juke 1989). While it is implied by MacArthur that dermatitis was not a symptom noted by Dr. Reid for the patients diagnosed with typhoid (he suggests it could have been typhus without a rash), perhaps these patients did not develop erythema because they were not exposed to sunlight in the workhouse

or the hospital (MacArthur 1956). While there is still the case that diarrhea was not recorded as present in these patients, pellagra should be considered as a possible cause for the other symptoms.

There is no mention of a disease that affects the tongue in the 1851 Census and people who experienced this symptom would likely have been diagnosed with typhoid and placed in the fever category. It is important to note that many of the deaths that occurred in the Kilkenny Union Workhouse are not accounted for in the census due to the poor preservation of records. Therefore, some diseases that were certain to have existed and would likely have caused many deaths are not documented. Scurvy, for example, would have been prevalent outside and inside the workhouse due to the absence of Vitamin C in a maize heavy or nutritionally vacant diet and pathological lesions indicative of scurvy are frequent in the skeletons recovered from the workhouse as described earlier (Geber 2015).

In the past, pellagra was frequently misdiagnosed as scurvy before the disease was better understood (Harris 1919; Brenton and Paine 2007; Ginnaio 2011) and is likely still misdiagnosed in skeletal assemblages from archaeological populations that experienced general malnutrition. Skeletal characteristics of pellagra were originally noted in earlier studies that describe osteoporosis and the thinning of the cortical bone in ribs, long bones, and phalanges (Lombroso 1892; Marie 1910; Roberts, 1914) but Brenton and Paine observed many similarities in lesion presence and histomorphometry between individuals who died with pellagra and those who died with scurvy and/or iron deficiency anemia, including the observation of cranial pitting, alveolar resorption, dental caries, extreme cortical loss, and lower bone turnover rates (Brenton and Paine 2007). Since those who were buried at the Kilkenny Union Workhouse were experiencing long-term malnutrition due to their social circumstances during the Great Famine, it is likely many people suffered from a combination of pellagra and scurvy.

Studies on the macroscopic presentation of pellagra in skeletal remains are limited, so it is unknown whether pellagra exhibits pathognomonic osseous changes; therefore, historical and cultural contextualization is crucial for determining whether the people who make up skeletal assemblages suffered from maize dependent diets. However, carbon isotope analysis of bone

collagen is another line of evidence that can indicate whether maize was a primary component of a person's diet in the years before their death.

In the Kilkenny Union Workhouse sample, many of the individuals with C_3 values are diagnosed as having scurvy and most of the individuals who exhibited high C_4 values through isotope analysis also exhibited macroscopic evidence of scurvy. This complicates the picture of the effect of diet on the presence of scurvy, but the introduction of bone histological analysis may be able to assist since differences were observed between the osteon size in those with varying $\delta^{13}C$ values where those with indications of maize showed increased Haversian canal and osteon size indicating more immature bone, while individuals with scurvy appear to have more mature bone properties such as decreased osteon area.

6.4. CHAPTER SUMMARY

The results discussed in this chapter indicate there is diversity in bone health, which reflects overall health status within the Kilkenny Union Workhouse sample. This is based on the variation in rib histomorphometry observed between the types of disease expressed in the skeletons those who were buried there. Dietary stable isotopes also show a relationship with bone histomorphometry which may indicate bone health was impacted by the presence of relief food. While some disease types affected bone remodeling in a more significant way than others, previous life circumstances and social standing which dictated access to resources outside and within the workhouse likely played a major role in the variation in health status within this sample. These results indicate there is room for further exploration of the link between bone histology, macroscopic bone lesions, and stable isotope values to better understand the risk of death in the Kilkenny Union Workhouse population and other archaeological samples in paleopathological studies.

CHAPTER 7. CONCLUSION

This goal of this thesis was to use histological methods to determine how disease and diet affected the health of a population of people who suffered during the Great Famine in mid-nineteenth century Ireland. In this analysis, the impact of poor living conditions, a monoculture system, and a history of resource deprivation was examined through the bone histology of the rib using an intra-site study of those who were buried on the grounds of the former Kilkenny Union Workhouse in Kilkenny, City, Ireland. This was achieved by comparing disease types identified through paleopathological analysis and variations in diet as observed in carbon and nitrogen stable isotopes with bone histomorphometric variables.

The cortical bone in the ribs of people who were buried on the Kilkenny Union Workhouse grounds demonstrates there are differences between disease types for some histomorphometric variables. Overall, rib bone porosity is the histomorphometric variable most affected by disease types, however there are associations between disease type and osteon area and correlations between diet and osteon and Haversian canal area. Each of these variables are known to be influenced by metabolic changes in the body. The presence of lesions indicative of infectious disease has a particularly strong effect on bone density where adults with evidence of infectious disease have less porosity than those without infectious disease and without lesions. Those with combined metabolic and infectious disease have the most dense bone while those without lesions have the most porosity and the most immature remodeling patterns. In subadults the trend is similar in that those with infectious disease have less porosity than those without disease, those with metabolic and infectious disease maintained the most dense bone, and those without lesions have the most porosity. However, these results are complicated by the fact that porosity is a common feature of cortical bone in young children and the presence of porosity in the youngest groups may be a result of combined malnutrition, disease, and factors related to growth and development.

The prevalence of individuals with lesions indicative of scurvy within this population indicates Vitamin C deficiency had a major impact on the victims of the Great Famine. The meaning of the presence of scurvy lesions is complicated and the absence of lesions in about half of the sample does not mean that these individuals were not suffering from scurvy. However, those

with scorbutic lesions appear to have healthier bone remodeling patterns than those without scurvy, which supports Geber's interpretation of resilience in older individuals with scurvy compared to younger individuals who died without evidence of scurvy (Geber 2015). In fact, the high amount of rib porosity in those without lesions relative to the rest of the disease types indicates disease processes were likely occurring that never presented on the surface of the bone.

Since scurvy can only be assessed skeletally after Vitamin C has been introduced back into the diet, the great number of people diagnosed with scurvy in the Kilkenny Union Workhouse skeletons is likely indicative of conditions outside the workhouse and the possibility that once people entered the institution, they were relieved of some of their nutritional deficiency (Geber and Murphy 2012; Geber 2015). However, heterogeneity in frailty suggests the state of a person's condition upon entry would dictate whether they lived long enough to show evidence of their suffering after death. Despite the strong evidence for scurvy in the skeletal record, Geber notes the lack of historical documentation of the disease in the records from the time (Geber 2015). This is likely due to the similarities of clinical symptoms between scurvy, pellagra, and infectious diseases classified as "famine fever". It is possible people suffered from both scurvy and pellagra as well as other comorbidities that cause a decline in cortical area and increase in cortical porosity for those with pathological lesions indicative of metabolic disease. However, those without lesions also appear to have the least healthy bone microstructure of all the disease groups. If this group is considered the most frail based on the appearance of bone histology alone, then this is evidence in support of the osteological paradox, which suggests those without lesions were the most vulnerable (Wood et al. 1992).

The issue of interpreting diet through the evaluation of stable isotopes from a population of nutritionally starved individuals is inherently linked to the histomorphometric variables assessed in this study. While Beaumont and colleagues observed a link between the C_4 levels present in the rib bone collagen of the Kilkenny Union Workhouse sample, the incorporation of carbon and nitrogen isotopes into the bone tissue of these individuals is only possible if collagen is being synthesized and incorporated into the bone matrix. As with the analysis of bone histology, isotope analysis is a retrospective look into the diets of those who died and may

reflect a period of years before death, therefore it is tempting to assume that those who show evidence of maize in their diet lived longer than those who do not. However, maize was not the only relief food provided during the Great Famine and access to soup kitchens and the minimal diversity known to be present in the stir-about sometimes provided there or within the workhouses will also be reflected in these tissues. In this study, children with indications of maize in their rib bone isotopes show evidence of less mature remodeling than those with isotopic values more closely aligned with C_3 foods. Since the study of effects of disease and diet on subadult bone microstructure is limited and studies on "normal" variation in subadult bone are lacking, it is not possible to say whether the presence of maize had a negative effect on the remodeling process of subadult bone, though this hypothesis would support the research by Brenton and Paine who showed that adults with pellagra are often underaged (Brenton and Paine 2007). While their research was focused on adults, it is worth considering whether the presence of secondary osteons in individuals as young as one and two years old in this sample indicates an acceleration in remodeling in order to increase calcium availability or protection against fracture, which may not have been possible for those who only had access to maize. Perhaps the presence of scorbutic lesions in this subadult sample indicates a re-introduction of Vitamin C that allowed for the rapid deposition and mineralization of the osteoid matrix that would have been required to initiate secondary osteon formation in these young children. It is possible the presence of isotopic values indicative of maize consumption is representative of a group of children who were not provided supplementary Vitamin C and, therefore, did not experience the same increase in the rate of bone remodeling. These are some of the many questions that were not answered in this thesis and deserve further exploration.

Additionally, the impact of maize as a relief food and the likelihood that many people in the workhouse suffered from pellagra is worthy of more in-depth analysis. While maize can provide some nutrition, particularly niacin when properly processed, the rapid and inefficient distribution of the foreign food initially prevented the poor from obtaining any vitamins as many people did not know how to consume the food and suffered from intestinal issues. Many of these people would already have been suffering from pellagra as they were receiving little to no food relief at all and the introduction of maize would not have helped their situation until they learned how to properly cook and consume the food.

Evidence of maize was less likely to show in adults due to the slower rate of bone turnover and the likely brief period of time between access to maize and death in this population. However, some adults in this sample do show isotopic values in the C_4 range and the study of incremental analyses of dentine from the same adults by Beaumont and Montgomery supports the transition from C_3 crops (potatoes) to a diet composed of food in the C_4 range, presumably maize (Beaumont and Montgomery 2016). This study showed a rise in osteon area, a variable correlated with cortical rib bone maturity, with the rise in $\delta^{15}N$ values, which is often associated with nutrient deficiency when protein is known to be absent from the diet. Whether maize was present in their diets or not, those who were buried in this cemetery were not receiving enough nutrients from the food to support an immune system that would protect them through the poorest of living conditions, the multiple periods of starvation, and the rampant disease they were regularly exposed to.

Colin Sage, a professor of geography at the University College Cork has discussed modern food poverty, a measure of social deprivation, as a consequence of three determinants (Sage 2012): The first is the lack of sufficient funds, an issue for those who experience food scarcity and simply do not have the monetary resources to purchase healthy food. The second determinant is the location of access to healthy food, which are often outside the city centers and not in the regions of what are now called "food deserts"— the inner cities or in poor rural areas where people are surviving on nutrient poor pre-processed foods. The third is access to food preparation knowledge and the ability to obtain nutrients from fresh food through proper cooking methods (Sage 2012). While these are modern examples of the issues faced by people who live in poverty today, they were also experienced by those living prior to the Great Famine. The reliance of the poor on the potato often meant that money was not a part of the exchange necessary for them to obtain enough nutrients to survive, so when the potato became scarce and the price of the crop increased the poor did not have funds to purchase supplementary food items. The distribution of maize was an additional consequence of this deprivation as it was cheap or sometimes free for those who were destitute but, as discussed in Chapter 3, it did not offer much nutritional value and therefore was not an adequate substitute for the potato. This is exemplified in the frequency of macro- and microscopic evidence of poor health described in

this thesis, where the prevalence of lesions indicative of disease and the lack of skeletal maturity and bone density indicate long-term struggles with maintenance of essential nutrients. In essence, the distribution of maize to the starving population of Ireland was the historic equivalent of the practice of making nutrient poor foods the only available resource to those who suffer in poverty today.

Structural violence is a useful framework for understanding the physiological effects of wealth disparities in a hierarchical society such as the one that existed between England and Ireland during the Great Famine and those that dominate the world today. Structural violence is commonly exercised on people in the lower strata of society by individuals in the higher strata, usually in the form of physical actions that cause harm or death as a means of social control (Farmer 2004; Scheper-Hughes 2004). However, Klaus argues that structural violence can be performed by more subtle methods, such as the restriction of resources like food and housing, that led to skeletal indicators of stress, and perhaps even more damaging, long-term consequences of physical and mental health through weakened immunocompetence and altered epigenetic expression of genes (Klaus 2012).

This discrete performance of structural violence is more difficult to recognize and the perpetrator is more difficult to pinpoint—particularly since this form of violence exists in a capitalist society where people on the lower rungs are expected to take responsibility for their place on the ladder. No matter the nomenclature nor the intent—workhouses, poorhouses, almshouses, detention centers—government-sponsored facilities designed to house the poor, the destitute, the asylum seekers, the criminally convicted are only as helpful as the governments who support them and those who inhabit them.

Galtung argues that the harm of structural violence can be blanketed through cultural violence, a form of indirect violence that functions to normalize discrimination and encourage assimilation through the ridicule and decimation of cultural practices like religion and language (Galtung 1990). This practice isolates, alienates, and strips the agency from those who end up in institutions like the workhouses. For Ireland, the Act of Union was the form of cultural violence that allowed Britain to have control over the response to the Great Famine. This long

history of discrimination dating back to the Penal Laws, which restricted the rights of native Irish Catholics, played a role in the development of a monoculture and the unstable practice of land subdivision (Smyth 2012). As a result, when the potato failed, the long-withheld restriction of access to resources, the lack of personal property, and the inability to accumulate wealth led to the workhouses, the decimation of thousands of homesteads (bothán), and the death of one million people.

This thesis described how bone histological analysis may help interpret frailty in skeletal assemblages from archaeological sites and aid in the understanding of how diet impacts bone health through multidisciplinary methods. More importantly, it provided new information about the experience of adults and children who suffered from poverty during the Great Famine through an investigation of their bone histology. The metabolic and infectious diseases that thrived during the Great Famine were deadly consequences of the structural violence experienced by the poor in mid-nineteenth century Ireland. While the circumstances may be different, structural violence is a continuous paradigm in hierarchical societies and still causes physiological and psychosocial stress among the most vulnerable today (Beatrice and Soler 2016; Sabo 2014; Suarez-Bregua et al. 2018). This is evident in recent forensic skeletal analyses found significantly more indicators of non-specific stress, including porotic hyperostosis, enamel hypoplasias, and double zonal osteons in people who died when crossing the border from Mexico into Arizona and Texas than in American born samples (Beatrice and Soler 2016; Goots et al. 2017).

In the future, paleopathologists who are able to gain permissions for destructive analyses should consider using cortical bone histology in addition to the examination of skeletal lesions to aid in interpretations of population health. If destructive analysis is not possible, Micro-CT can provide information about cortical area and percent cortical area, which were shown to be influenced by sex and disease type in this study. The presence of larger cortices in females in the Kilkenny Union Workhouse population was unexpected and studies of other marginalized historic populations should consider using bone histology to investigate the female buffering hypothesis (Stinson 1985). Additionally, future research should seek to include bone histological analysis of subadult samples to more accurately identify the effective age of adult

compacta and determine the association between health and secondary osteon accumulation in children. This research is warranted given the large number of secondary osteons that are present in very young individuals under the age of twelve in this sample. Finally, bone histologists who conduct age at death estimation for modern and historic populations should be aware of the impact of disease and diet on bone histomorphometry in groups that are currently or have experienced structural violence. Since individuals who are unidentified in forensic contexts are often a part of marginalized groups with less access to beneficial resources (Paine and Brenton 2007), anthropologists should exercise caution when using bone histology methods for estimation age at death. The next step in this research is to use histological methods for age-at-death estimation of the Kilkenny Union Workhouse population and compare those results to the estimates obtained by Geber (2015) using gross macroscopic methods. The results of that study will reveal more about the relationship between age associated variables in bone histology and the limitations of both macroscopic and microscopic methods for age estimation.

The recent historical and biological investigations into the life experience of the people who died at the Kilkenny Union Workhouse shows the impact of forces acting both within and outside the body that contributed to the poor state of Irish well-being prior to and throughout the Great Famine. The impact of the laissez-faire attitudes of government officials who ignored signs of economic failure and then restricted access to substantial resources at the height of the worst famine in Irish history, are reflected in the number of people who died and the number of people who left the country, but it can also be observed in the skeletons of those who perished in Kilkenny City. The analysis of microscopic changes to the skeleton in response to malnutrition and disease presented in this thesis reinforces the massive suffering that occurred within the confines of the Kilkenny Union Workhouse institution and throughout workhouses all over Ireland.

Figure 7.1. A monument dedicated to those who perished in the Great Famine and other famines around the world. Photographed by the author in the Callan Famine Cemetery in County Kilkenny, Ireland.

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APPENDICES

Appendix 1.1. OHI and BI values for adults from the Kilkenny Union Workhouse population sample

ID	OHI	BI
2	0	0
5	0	0
13	3	1
13	4	1
14	4	1
16	0	0
17	0	0
20	1	0
23	1	0
26	2	0
35	0	0
48	1	0.5
53	5	1
75	1	0.5
79	5	1
85	0	0
87	0	0
90	2	0.5
92	2	0
94	4	0.5
97	4	1
105	5	1
108	3	0.5
116	2	0
117	4	1
126	4	1
127	5	1
128	5	1
133	4	1
138	2	0.5
139	1	0.5
141	5	1
155	0	0
158	5	1
167	0	0
172	3	0.5
174	5	1

175	5	1
ID	**OHI**	**BI**
179	4	1
190	0	0.5
195	1	0.5
205	4	1
215	1	0
216	1	0
219	5	1
220	1	0.5
221	4	1
222	4	1
224	2	0.5
227	4	1
233	5	1
234	3	1
241	0	0
246	5	1
255	4	1
257	0	0
269	4	1
271	4	1
279	0	0
282	5	1
285	5	1
289	4	1
297	3	1
298	5	1
299	5	1
308	5	1
316	1	0
317	5	1
318	2	0
320	5	0.5
321	3	1
333	2	0.5
342	4	1
343	5	1
346	0	0
347	5	1
356	3	1

366	1	0
ID	**OHI**	**BI**
367	1	
378	1	0
385	4	1
387	0	0
396	5	1
398	5	1
399	1	0
401	0	0
404	3	1
407	5	1
416	0	0
417	3	0.5
422	1	0
424	0	0.5
428	4	0.5
433	5	1
437	1	0
440	3	1
446	2	0
448	1	0
451	5	1
456		
456	3	0.5
474	0	0
486	4	1
491	0	0
495	5	1
508	5	1
520	3	1
521	4	0.5
531	4	0.5
543	4	1
551	5	1
556	4	1
557	5	1
559	5	1
560	5	1
564	5	1
572	5	1

574	5	1
ID	**OHI**	**BI**
575	3	0.5
576	5	1
577	0	0
582	4	0.5
583	5	1
597	5	1
598	5	1
608	5	1
611	5	1
611	5	1
615	1	0.5
629	5	1
655	5	1
661	5	1
678	0	0
681	5	0.5
682	0	0
683	5	1
685	5	1
693	5	1
695	4	1
697	0	0
711	1	0
736	5	1
737	3	1
737	3	1
744	1	0
745	5	1
757	5	1
765	0	0
772	5	1
772	5	1
783	5	1
785	4	1
787	4	1
788	5	1
789	5	1
796	4	1
800	4	1

802	5	1
ID	**OHI**	**BI**
804	5	1
808	5	1
818	5	1
826	1	0
828	1	0
829	1	0
835	1	0
844	5	1
855	5	1
856	5	0.5

Appendix 1.2. OHI and BI values for subadults from the Kilkenny Union Workhouse population sample

ID	OHI	BI
11	5	1
15	0	0
19	5	1
24	2	0.5
25	1	0
29	0	0
31	2	0
33	1	0
36	0	0
40	0	0
42	2	0.5
44	2	0
45	0	0
49	0	0
73	0	0
76	3	0.5
77	0	0
106	2	0
107	0	0
112	1	0
113	1	0
132	5	1
135	0	0
136	4	0.5
151	5	1
152	5	1
153	3	0.5
154	1	0
159	5	1
173	4	1
180	2	0.5
184	4	1
185	0	0
187	1	0.5
193	5	1
204	2	0
207	4	0.5
211	5	1
214	1	0.5

IND	OHI	BI
223	5	1
228	2	0.5
230	5	1
235	5	1
237	4	1
251	1	0
253	5	1
258	5	1
262	4	1
283	3	0.5
292	4	1
296	5	1
305	5	1
336	5	1
345	0	0
348	5	1
354	1	0
355	3	0.5
358	1	0
361	5	1
369	5	1
372	4	1
373	3	0.5
377	4	1
381	5	1
388	5	1
418	5	1
419	5	1
420	4	1
421	4	1
423	5	1
431	3	1
434	0	0
436	4	1
442	4	1
444	5	1
453	4	1
464	5	1
464	5	1
493	4	1

IND	OHI	BI
496	5	1
500	1	0
501	5	1
502	5	1
513	4	1
525	5	1
526	5	1
528	3	1
532	5	1
538	5	1
539	5	1
542	3	0.5
545	5	1
548	4	1
549	5	1
553	5	1
563	5	1
585	4	1
591	5	1
593	5	1
603	5	1
609	5	1
612	5	1
614	5	1
621	0	0
624	5	1
626	5	1
636	5	1
658	4	0.5
666	1	0
670	5	1
679	3	0.5
684	3	1
687	3	0.5
707	5	1
715	5	1
718	4	1
719	5	1
760	5	1
770	3	0.5

IND	OHI	BI
777	5	1
778	4	1
784	5	1
793	4	1
794	4	1
801	5	1
830	0	0
836	0	0

Appendix 1.3. Age, sex, disease, and histomorphometric data for adults in the Kilkenny Union Workhouse population sample

IND	Sex	Age (yrs)	Scurvy	Disease	% Ct_Ar	On.Ar	On.Cr	Ha.Ar	HaCr	Po.Ar	% PoAr	DZ	OPD
13	M	28.3	No	None	x	0.06	0.90	0.0021	0.88	x	x	x	x
14	F	24.7	No	None	x	0.04	0.90	0.0009	0.93	x	x	x	x
53	F	20.5	No	None	x	0.04	0.84	0.0039	0.87	x	x	x	x
79	F	48.6	No	M	36.67	0.03	0.90	0.0018	0.90	1.49	6.48	x	13.7
94	F	52.7	No	M	44.51	0.05	0.87	0.0014	0.91	1.11	4.96	4	8.7
97	F	39.8	No	None	x	0.04	0.87	0.0019	0.89	x	x	x	x
105	F	31.4	No	None	32.90	0.02	0.90	0.0012	0.91	0.09	0.68	x	x
117	F	39.2	No	None	37.93	0.03	0.88	0.0011	0.94	0.62	4.00	13	23.0
126	F	30.0	No	M	x	0.07	0.88	0.0014	0.91	0.42	1.43	20	11.3
127	M	35.0	Yes	M	46.38	0.04	0.88	0.0053	0.86	2.35	6.57	4	10.3
128	M	43.9	No	M	50.79	0.03	0.90	0.0014	0.87	1.35	5.00	5	13.1
133	F	40.6	No	M&I	43.14	0.03	0.88	0.0007	0.94	0.28	1.38	4	16.3
141	M	51.5	Yes	M	32.55	0.03	0.92	0.0024	0.93	x	x	6	13.4
158	F	36.3	No	None	x	0.04	0.89	0.0016	0.89	1.24	x	x	x
175	M	34.8	Yes	M	28.70	0.04	0.90	0.0014	0.92	0.51	2.35	x	8.9
179	F	35.6	Yes	M&I	x	0.03	0.87	0.0011	0.95	x	x	x	x
205	F	35.1	No	None	x	0.04	0.87	0.0015	0.95	x	x	x	x
219	M	41.6	Yes	M	27.98	0.04	0.92	0.0020	0.88	0.48	2.04	3	16.9
221	M	42.5	Yes	M	x	0.03	0.86	0.0014	0.92	x	x	x	x
222	F	35.1	No	None	30.68	0.03	0.89	0.0009	0.94	0.22	1.79	x	x
227	M	51.8	Yes	M	21.89	0.01	0.91	0.0013	0.95	0.27	1.97	2	11.8
233	M	44.3	Yes	M&I	33.25	0.03	0.93	0.0017	0.92	0.47	2.53	4	13.1
234	F	34.2	No	M	x	0.05	0.90	0.0017	0.90	x	x	x	x
255	M	42.1	No	None	x	0.03	0.91	0.0013	0.97	x	x	x	x
269	F	25.7	Yes	M	41.14	0.04	0.88	0.0011	0.94	0.41	2.21	5	8.2
271	M	39.3	Yes	M	x	0.03	0.88	0.0012	0.90	x	x	9	15.2
282	M	44.3	Yes	M	31.18	0.03	0.89	0.0010	0.92	0.13	1.01	10	15.1
285	M	30.6	Yes	M	37.25	0.04	0.88	0.0015	0.92	x	x	1	14.5
289	F	28.9	No	None	65.67	0.06	0.86	0.0016	0.86	0.84	3.34	4	6.4
297	F	29.3	No	None	36.25	0.07	0.86	0.0014	0.88	x	x	x	x
298	M	46.3	No	None	x	0.03	0.88	0.0015	0.93	x	x	x	x
299	M	33.4	Yes	M&I	26.01	0.05	0.92	0.0018	0.94	x	x	10	13.0
308	M	22.8	Yes	M	47.62	0.04	0.90	0.0020	0.95	1.97	7.60	21	11.0
317	F	29.8	No	None	x	0.03	0.87	0.0010	0.90	x	x	x	x
320	F	28.9	Yes	M	54.01	0.04	0.88	0.0017	0.92	0.47	1.66	1	6.9
321	F	32.1	Yes	M&I	x	0.03	0.90	0.0012	0.94	x	x	x	x
342	M	45.4	Yes	M&I	x	0.04	0.91	0.0016	0.95	x	x	10	18.4
343	M	21.5	No	M	43.76	0.05	0.84	0.0016	0.91	1.20	3.33	x	x

IND	Sex	Age (yrs)	Scurvy	Disease	% Ct_Ar	On.Ar	On.Cr	Ha.Ar	HaCr	Po.Ar	% PoAr	DZ	OPD
347	M	35.7	Yes	M	x	0.03	0.89	0.0011	0.95	x	x	x	x
356	F	31.6	Yes	M	x	0.04	0.85	0.0009	0.93	x	x	x	x
385	F	39.4	No	I	31.41	0.04	0.87	0.0009	0.96	1.13	6.66	0	19.0
396	M	30.2	Yes	M&I	x	0.04	0.87	0.0013	0.97	0.53	x	0	7.9
398	M	38.3	No	None	21.73	0.02	0.90	0.0012	0.91	0.53	2.81	3	18.1
404	F	52.0	No	I	35.64	0.04	0.88	0.0009	0.91	0.82	3.78	x	x
407	F	39.9	Yes	M&I	37.89	0.04	0.92	0.0016	0.93	0.44	3.09	0	15.1
417	M	25.9	No	M	42.30	0.05	0.86	0.0017	0.89	0.60	2.40	x	x
428	F	41.0	No	M	x	0.05	0.87	0.0012	0.90	x	x	x	x
433	F	35.1	Yes	M&I	x	0.04	0.89	0.0012	0.88	x	x	x	x
440	M	47.1	Yes	M	x	0.05	0.87	0.0015	0.91	x	x	x	x
451	F	39.6	No	I	41.35	0.04	0.92	0.0015	0.95	x	x	9	17.6
486	M	50.6	Yes	M	26.39	0.05	0.88	0.0010	0.94	0.31	1.60	6	15.3
495	F	25.7	No	M&I	58.05	0.05	0.88	0.0013	0.92	0.51	1.87	2	9.1
508	M	24.6	No	None	x	0.04	0.87	0.0016	0.95	0.15	x	2	3.9
520	F	38.2	No	I	x	0.04	0.92	0.0011	0.92	0.47	x	2	x
521	M	33.3	No	None	x	0.04	0.83	0.0015	0.88	x	x	x	x
531	M	42.2	Yes	M&I	x	0.03	0.88	0.0011	0.94	x	x	x	x
543	F	41.7	Yes	M	37.01	0.03	0.90	0.0017	0.96	x	x	x	12.6
551	F	41.7	No	None	35.29	0.04	0.89	0.0010	0.96	0.81	5.33	3	10.7
556	F	24.7	No	None	x	0.05	0.92	0.0011	0.85	x	x	8	13.3
557	F	38.5	No	I	33.13	0.03	0.90	0.0062	0.90	0.50	3.47	2	13.5
559	F	34.6	Yes	M	26.61	0.03	0.89	0.0012	0.96	0.19	1.72	1	13.7
560	M	22.3	Yes	M	37.39	0.04	0.87	0.0016	0.92	0.39	2.29	3	14.0
564	F	25.9	No	I	45.96	0.04	0.90	0.0009	0.94	0.16	0.98	2	11.4
572	F	45.2	Yes	M	49.26	0.04	0.92	0.0014	0.96	1.63	7.54	2	15.4
574	M	45.4	No	None	x	0.03	0.87	0.0015	0.94	x	x	x	x
576	F	33.6	No	None	46.18	0.04	0.88	0.0010	0.93	x	x	x	x
582	F	25.7	No	None	39.52	0.04	0.88	0.0011	0.92	0.94	4.88	8	21.5
583	F	43.8	Yes	M	x	0.03	0.88	0.0013	0.95	1.02	x	x	x
597	M	41.8	Yes	M	28.70	0.03	0.90	0.0013	0.92	0.20	1.26	7	16.1
598	M	33.7	Yes	M	42.49	0.05	0.86	0.0014	0.93	0.81	3.09	3	15.6
608	M	51.6	Yes	M	25.38	0.04	0.91	0.0013	0.95	0.90	x	4	13.7
611	M	37.7	Yes	M&I	29.80	0.04	0.87	0.0014	0.89	0.34	1.99	1	11.6
629	F	28.5	No	M	69.18	0.04	0.88	0.0011	0.95	x	1.71	4	7.1
655	F	28.1	Yes	M	51.84	0.04	0.89	0.0013	0.95	x	x	5	11.3
661	F	55.9	No	None	28.06	0.03	0.91	0.0010	0.97	0.83	5.95	3	15.7
681	F	24.5	No	None	52.23	0.04	0.88	0.0011	0.93	0.21	1.02	3	9.5
683	M	45.0	No	I	50.14	0.05	0.93	0.0017	0.97	0.66	1.98	5	13.6
685	F	43.3	No	None	52.11	0.05	0.89	0.0019	0.94	0.28	5.87	0	12.9

IND	Sex	Age (yrs)	Scurvy	Disease	% Ct_Ar	On.Ar	On.Cr	Ha.Ar	HaCr	Po.Ar	% PoAr	DZ	OPD
695	F	34.9	Yes	M	51.40	0.03	0.90	0.0008	0.94	0.35	1.62	2	9.1
736	M	37.9	No	I	x	0.03	0.90	0.0007	0.95	x	x	2	2.9
737	M	42.1	No	M	x	0.05	0.88	0.0018	0.92	x	x	x	x
745	M	27.7	Yes	M	37.62	0.03	0.89	0.0007	0.96	0.34	1.80	3	19.4
757	F	37.1	No	None	36.23	0.04	0.88	0.0012	0.95	x	x	x	10.9
772	F	42.8	No	None	x	0.03	0.89	0.0016	0.94	x	x	x	x
783	M	33.2	Yes	M	40.36	0.05	0.90	0.0013	0.96	x	x	6	11.4
785	M	37.2	Yes	M	34.74	0.04	0.90	0.0014	0.95	0.54	2.13	7	15.7
787	M	43.4	No	None	x	0.05	0.92	0.0009	0.95	x	x	x	x
788	M	44.5	No	None	x	0.04	0.78	0.0009	0.91	0.96	x	10	8.8
789	F	38.9	No	None	48.31	0.04	0.86	0.0051	0.92	1.21	5.99	1	11.0
796	F	31.1	Yes	M	35.68	0.05	0.89	0.0020	0.94	0.73	x	0	14.1
800	F	43.0	Yes	M	x	0.04	0.87	0.0013	0.91	x	x	x	x
802	F	34.5	Yes	M	29.79	0.05	0.90	0.0015	0.94	x	1.89	5	13.1
804	M	50.5	No	None	x	0.04	0.90	0.0011	0.94	x	x	x	x
808	M	35.1	Yes	None	36.08	0.06	0.85	0.0012	0.94	0.74	3.33	3	11.0
818	F	33.4	No	M	47.89	0.04	0.88	0.0014	0.95	1.27	7.30	1	11.0
844	F	32.4	No	I	64.19	0.04	0.84	0.0012	0.89	0.38	1.30	1	10.8
855	M	30.5	No	None	x	0.05	0.88	0.0014	0.89	x	x	x	x
856	F	53.7	No	None	x	0.03	0.92	0.0010	0.98	x	x	x	x

Appendix 1.4. Age, sex, disease, and histomorphometric data for subadults in the Kilkenny Union Workhouse population sample

IND	Sex	Age	Scurvy	Disease	% Ct_Ar	On.Ar	On.Cr	Ha.Ar	HaCr	Po.Ar	% PoAr	OPD
19	Ind.	4.0	Yes	M	54.25	0.04	0.85	0.00	0.91	1.27	x	x
132	Ind.	9.0	Yes	None	82.85	0.04	0.85	0.00	0.93	1.25	5.86	2.30
135	Ind.	1.3	Yes	M	x	0.06	0.82	0.00	0.86	x	x	x
136	Ind.	16.5	Yes	M&I	x	0.03	0.88	0.00	0.91	x	x	x
151	Ind.	4.0	Yes	M	x	0.03	0.87	0.00	0.92	x	x	x
152	Ind.	6.0	Yes	M	55.55	0.03	0.90	0.00	0.89	0.44	3.10	3.95
153	Ind.	13.5	Yes	M&I	x	0.06	0.82	0.00	0.86	0.29	x	x
159	Ind.	13.0	Yes	M	56.02	0.04	0.89	0.00	0.94	0.65	2.83	3.98
173	Ind.	7.0	Yes	M	38.62	0.03	0.87	0.00	0.83	0.35	4.04	1.16
184	Ind.	3.5	Yes	M	57.00	0.04	0.80	0.00	0.93	0.78	7.38	1.22
193	Ind.	1.5	Yes	M	55.22	0.03	0.85	0.00	0.87	2.27	21.61	0.38
207	Ind.	13.0	Yes	M	x	0.05	0.85	0.00	0.92	1.54	6.89	0.85
211	Ind.	3.0	No	M	36.18	x	x	x	x	0.08	0.80	0.00
223	Ind.	10.0	Yes	None	47.15	0.04	0.89	0.00	0.90	1.01	5.53	2.09
230	Ind.	6.5	Yes	M	37.83	0.04	0.80	0.00	0.90	0.66	5.15	1.79
235	Ind.	9.0	Yes	M&I	x	0.03	0.88	0.00	0.92	0.62	4.70	4.25
237	Ind.	17.5	Yes	M	38.42	0.04	0.87	0.00	0.91	0.55	3.02	2.98
253	Ind.	7.0	Yes	None	48.95	0.04	0.87	0.00	0.91	1.61	10.03	2.50
258	Ind.	6.5	Yes	M	58.84	0.04	0.87	0.00	0.88	1.30	8.73	2.21
262	Ind.	5.0	Yes	M	x	0.03	0.90	0.00	0.95	x	x	x
292	Ind.	2.0	Yes	M	31.93	0.02	0.89	0.00	0.96	0.16	1.42	0.71
296	Ind.	2.5	Yes	M	x	0.05	0.80	0.00	0.87	x	x	x
305	Ind.	3.5	Yes	M	x	0.02	0.87	0.00	0.91	0.07	x	x
336	Ind.	3.0	Yes	M	47.44	x	0.90	x	0.92	2.23	5.96	0.48
348	Ind.	5.0	Yes	M	52.47	x	0.87	x	0.87	3.84	5.46	0.20
355	M	16.0	No	M	59.72	0.05	0.91	0.00	0.87	x	9.07	0.21
361	Ind.	13.0	No	M&I	57.01	0.04	0.87	0.00	0.87	0.83	4.76	1.08
369	Ind.	2.8	Yes	None	33.91	0.02	0.86	0.00	0.93	x	2.74	0.62
372	Ind.	12.0	Yes	M	x	0.04	0.86	0.00	0.89	0.42	x	x
373	Ind.	16.5	No	M	41.52	0.06	0.89	0.00	0.91	2.18	9.43	1.47
377	Ind.	6.0	Yes	M	41.26	0.04	0.84	0.00	0.94	1.76	15.21	1.82
381	Ind.	3.0	Yes	M	41.93	0.03	0.88	0.00	0.90	0.57	5.39	4.00
388	Ind.	6.0	No	M	48.59	0.04	0.83	0.00	0.92	0.97	8.19	1.60
418	Ind.	5.5	Yes	M	40.37	0.03	0.91	0.00	0.87	0.77	6.28	1.31
419	Ind.	5.0	Yes	M	44.33	0.02	0.86	0.00	0.89	0.57	5.92	1.36
420	Ind.	4.0	Yes	M	x	0.02	0.92	0.00	0.93	0.77	x	x
421	Ind.	6.0	Yes	None	57.40	0.04	0.81	0.00	0.87	1.67	9.75	2.10
423	Ind.	15.5	No	M	54.18	0.05	0.88	0.00	0.94	1.39	5.02	0.87

IND	Sex	Age	Scurvy	Disease	% Ct_Ar	On.Ar	On.Cr	Ha.Ar	HaCr	Po.Ar	% PoAr	OPD
431	F	15.0	No	I	58.82	0.04	0.89	0.00	0.91	x	3.18	0.77
436	Ind.	11.0	Yes	None	55.41	0.04	0.91	0.00	0.95	1.65	8.20	1.20
442	Ind.	10.5	Yes	M&I	58.30	0.05	0.87	0.00	0.90	1.31	5.48	2.14
444	Ind.	7.0	Yes	M	41.94	0.05	0.91	x	0.93	1.06	7.85	2.30
453	Ind.	14.0	No	M	x	0.04	0.92	0.00	0.95	x	x	x
464	Ind.	16.0	No	M	53.46	0.04	0.90	0.00	0.95	1.34	4.49	2.24
493	Ind.	9.5	Yes	M	52.36	0.04	0.91	0.00	0.93	1.66	x	0.88
496	F	15.0	Yes	M	53.80	0.04	0.87	0.00	0.93	1.49	4.35	2.40
501	Ind.	3.0	Yes	M	x	0.02	0.82	0.00	0.91	x	x	x
502	Ind.	5.5	No	M	42.18	0.04	0.85	0.00	0.93	x	2.83	2.01
513	Ind.	13.0	Yes	None	51.99	0.04	0.90	0.00	0.94	0.98	3.66	1.20
525	Ind.	11.5	No	M	44.57	0.04	0.89	0.00	0.95	1.76	8.12	2.27
526	Ind.	14.0	Yes	M	38.73	0.05	0.88	0.00	0.92	0.80	4.46	2.28
528	Ind.	4.0	Yes	M	57.33	0.05	0.85	0.00	0.97	0.60	3.87	1.36
532	Ind.	5.5	Yes	M	45.54	0.04	0.88	0.00	0.91	0.94	5.82	1.93
538	Ind.	13.0	Yes	None	54.68	0.05	0.88	0.00	0.92	1.02	6.63	3.20
539	Ind.	12.5	Yes	None	46.14	0.05	0.82	0.00	0.87	1.91	9.20	1.06
542	Ind.	7.0	Yes	M	53.70	0.06	0.90	0.00	0.92	1.22	7.16	1.94
545	Ind.	7.0	Yes	M	52.74	0.04	0.90	0.00	0.93	1.53	x	2.25
548	Ind.	7.0	No	M	x	0.04	0.88	0.00	0.91	0.02	x	x
549	Ind.	5.5	No	M	58.43	0.02	0.88	x	x	2.09	12.34	1.54
553	Ind.	12.0	No	None	55.54	0.04	0.86	0.00	0.87	0.95	5.35	3.73
563	Ind.	8.0	Yes	M	41.49	0.05	0.86	0.00	0.95	0.83	5.30	1.91
585	Ind.	2.0	Yes	M	46.73	0.03	0.90	0.00	0.90	0.92	7.34	2.31
591	M	17.0	No	M	29.72	0.05	0.86	0.00	0.92	3.05	12.74	2.88
593	Ind.	2.5	Yes	M	35.93	0.03	0.85	0.00	0.92	0.66	6.99	2.01
609	Ind.	11.5	Yes	M	59.54	0.07	0.90	0.00	0.90	1.03	3.80	2.92
612	Ind.	9.0	Yes	M	56.49	0.04	0.85	0.00	0.91	1.30	6.75	4.22
614	Ind.	15.5	Yes	M	42.03	0.03	0.87	0.00	0.91	1.57	5.76	2.31
624	Ind.	12.5	No	M&I	54.71	0.04	0.87	0.00	0.94	1.07	4.86	3.60
626	Ind.	7.0	Yes	M	x	0.03	0.90	0.00	0.91	0.73	4.27	1.40
636	Ind.	6.0	Yes	M	x	0.03	0.88	0.00	0.94	0.57	3.54	3.65
658	Ind.	6.0	Yes	M	x	0.02	0.87	0.00	0.94	x	x	x
670	Ind.	10.0	Yes	M&I	60.65	0.05	0.87	0.00	0.90	0.53	3.36	3.34
679	Ind.	7.0	No	M	48.80	0.04	0.89	0.00	0.93	0.81	4.35	0.97
684	Ind.	4.0	Yes	M	x	0.02	0.93	0.00	0.88	0.02	x	x
687	Ind.	11.0	Yes	None	x	0.04	0.89	0.00	0.92	x	x	x
707	Ind.	13.5	Yes	M	56.32	0.04	0.85	0.00	0.91	2.25	9.59	1.15
715	Ind.	9.0	No	None	52.55	0.04	0.89	0.00	0.90	1.28	7.52	3.61
718	Ind.	11.5	Yes	I	x	0.04	0.90	0.00	0.96	x	x	x

IND	Sex	Age	Scurvy	Disease	% Ct_Ar	On.Ar	On.Cr	Ha.Ar	HaCr	Po.Ar	% PoAr	OPD
719	Ind.	8.0	No	M	x	0.05	0.84	0.00	0.88	x	x	x
760	M	17.0	Yes	M	37.89	0.04	0.92	0.00	0.94	0.65	2.31	x
770	Ind.	8.0	Yes	M	x	0.04	0.87	0.00	0.86	x	x	x
777	Ind.	17.5	Yes	None	59.74	0.05	0.88	0.00	0.90	2.74	7.63	1.59
778	F	15.5	Yes	I	46.22	0.06	0.87	0.00	0.89	1.45	3.90	1.46
784	Ind.	10.5	No	None	x	0.05	0.88	0.00	0.90	x	x	x
793	Ind.	13.0	Yes	M	52.88	0.04	0.87	0.00	0.92	1.95	11.67	2.33
794	Ind.	5.0	Yes	None	39.18	0.04	0.88	0.00	0.96	0.87	7.95	2.74
801	Ind.	13.0	No	M	45.18	0.04	0.92	0.00	0.96	3.18	11.16	2.32

Appendix 1.5. Stable isotope values for adults from the Kilkenny Union Workhouse population sample. Obtained from Julia Beaumont (Beaumont et al. 2013)

Individual	$\delta^{13}C$	$\delta^{15}N$
14	-20.3	10.5
53	-19.6	10.5
495	-19.4	11.1
13	-17.2	11.6
695	-20.8	11.0
128	-20.0	10.5
158	-20.1	10.6
233	-18.8	10.8
398	-19.7	11.2
557	-19.9	11.0
572	-20.3	10.6
597	-18.9	11.4
611	-19.9	11.6
683	-20.2	11.6
79	-17.4	10.7
141	-19.7	10.2
227	-20.0	9.5

Appendix 1.6. Stable isotope values for subadults from the Kilkenny Union Workhouse population sample. Obtained from Julia Beaumont (Beaumont et al. 2013)

Individual	$\delta^{13}C$	$\delta^{15}N$
151	-15.1	9.6
418	-18.6	9.8
502	-17.0	10.1
532	-20.3	10.3
421	-16.6	8.7
444	-16.5	9.4
626	-18.1	9.4
636	-19.9	10.3
715	-17.3	10.6